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THE EFFECT OF INDUCED ASYSTOLE ON VENTRICULAR FUNCTION*

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UNDER the circumstances of cardio-pulmonary by-pass the heart beats vigorously and there is an appreciable return of blood to the chambers of the heart from the coronary and bronchial systems. These factors interfere with the precision of some of the manoeuvres involved in the repair of intracardiac defects. Since there is retrograde blood flow in the ascending aorta the application of a clamp across the aorta at this site prevents coronary blood flow, causing total myocardial ischaemia and a complete cessation of the coronary venous return to the right side of the heart. The heart continues to beat weakly for a few minutes and finally stops. Blood, however, continues to return to the left atrium through the anastomoses between the bronchial arteries and the pulmonary veins. This technique of simple aortic occlusion has been used to facilitate intra-cardiac surgery.

A more profound and immediate cessation of cardiac activity can be obtained by the injection of potassium citrate solution into the aorta proximal to the clamp so that it will perfuse the coronary vascular tree (Melrose, Dreyer, Bentall and Baker, 1955). The deliberate induction of asystole by the use of potassium citrate has been widely used in clinical practice (Effler, Groves, Somes and Kolff, 1956).

The experiments reported here were designed to study the effects of these procedures on the function of the heart. There were two reasons for the study. First, these procedures were freely adopted by surgeons before any proper objective evaluation of their effects was available and secondly, in our own practice, we had reason to believe that their use was not without danger.

Whilst prolonged cardio-pulmonary bypass itself results in no appreciable deterioration in ventricular function, incision of the ventricle invariably does (Stirling, Stanley and Lillehei, 1957). As it seemed probable therefore that any harmful effects of asystole would be additive to those of ventriculotomy these studies were carried out in dogs who were not subjected to ventriculotomy. In most other respects the technique used was similar to that described in 1957.

Method

The basic principle used was to subject a dog to a series of known volume loads before and after the interruption of its coronary blood flow during cardio-pulmonary by-pass. During these 2 test periods the pressure responses in the left atrium and the aorta were measured and the information obtained was used to calculate the work done by the left ventricle. The results were expressed as ventricular function curves (Sarnoff and Berglund, 1954) and related the minute work done by the left ventricle to the mean pressure in the left atrium. By comparing the control curve, obtained from observations made before by-pass and interruption of

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coronary blood flow, with that obtained after these events, some assessment of the effects on ventricular function could be made.

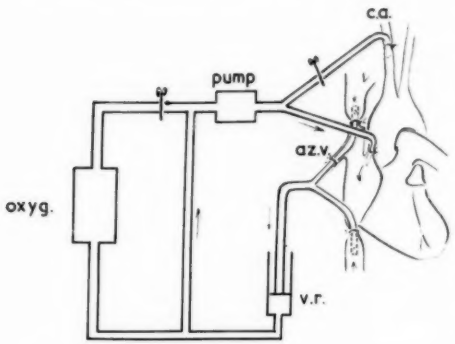

FIG. I. Schematic diagram of the experimental circuit. The venous return is siphoned from the 2 venae cavae to a reservoir (V.r.). The reservoir is emptied by a positive displacement pump which returns the blood to the right atrium. This is the circuit used for a work study; note that the efferent line from the oxygenator and the arterial lead to the carotid artery (c.a.) are clamped. During cardiac by-pass these clamps are removed and placed on (1) the line which short-circuits the oxygenator and (2) the lead to the right atrium.

Thirty-five mongrel dogs weighing between 10 and 20 kg. were used as subjects The animals were premedicated with morphine sulphate and atropine. Anaesthesia was induced with thiopentone and controlled respiration anaesthesia was continued using nitrous oxide and occasional supplements of thiopentone. A right thoracotomy was performed by excising the fifth rib. Tapes were passed around the superior and inferior venae cavae and threaded through rubber tubes to act later as occluding snares around the caval cannulae. Fine plastic cannulae were introduced through the femoral artery into the aorta and through a segmental pulmonary vein into the left atrium for continuous pressure measurement. In the early experiments these were connected to electromanometers but in later experiments simple mercury and saline manometers were used. Heparin (2 mg. per kg. body weight) was administered by the intravenous route and large cannulae were placed in the right common carotid artery, the superior vena cava, the inferior vena cava and the right atrium. The azygos vein was usually used for the superior vena caval cannula whilst the inferior vena caval cannula was introduced through the right atrial appendage in company with the right atrial cannula.

During a work study (Fig. I) the 2 caval cannulae were used to divert the whole of the systemic venous return through a circuit which included a collecting reservoir and a positive displacement pump[1, 2] which returned the blood unchanged to the right atrium.

The output of the pump was proportional to its rate of revolution so that, by reference to a tachometer, the cardiac inflow was known. The rate of inflow was changed in stepwise fashion and at each rate of flow the left atrial and aortic mean pressures were recorded. The flow rates used varied from a minimum of 60 ml. per kg. body weight per minute to a maximum of 150 ml. per kg. per min. The pressures and the volume in the extra-corporeal circuit stabilized in thirty to sixty seconds after change in flow. This information was used to construct the "control" ventricular function curve.

At this stage, by appropriate manipulation of clamps, the circuit was changed so that the venous blood collected by the caval cannulae gravitated into a spinning disc-oxygenator[3] where it was oxygenated and returned to the carotid artery at a flow-rate of 100 ml. per kg. per min.

After ten minutes of cardio-pulmonary by-pass asystole was induced by one or other of the techniques detailed below. During asystole the left atrium was decompressed by siphonage from the pulmonary vein cannula. At the end of asystole one of the caval snares was released to decompress the right heart and left atrial decompression was continued. Cardio pulmonary by-pass was continued for at least ten minutes after the heart had resumed its contractions and, after this time, the circuit for a ventricular function study was again used to collect data and construct a curve to be compared with the control curve. The following methods of producing asystole were used.

(1) The aorta was cross-clamped just below the origin of the brachio-cephalic artery and it was not released until the end of the period of occlusion had been reached. In 5

[1]Sigma Motor T.6S, Middleport, New Jersey.

[2]Mono Pump, Burwood, Victoria.

[3]Kay-Cross, Pemco Inc., Cleveland, Ohio.

animals this period was five minutes, in 3 it was ten minutes, in 5 it was twenty minutes and in 2 it was thirty minutes.

(2) The aorta was cross-clamped for a total period of twenty minutes but after each five-minute interval the aortic clamp was released for one minute.

(3) The aorta was cross-clamped for an uninterrupted period of twenty minutes, during this period the heart was liberally bathed in saline at 4°C so that the temperature in the right atrium was reduced to between 20°C and 27°C. The temperature invariably fell by 10°C in less than three minutes and was easily maintained in the above range by alteration of the rate of flow of the cold solution.

(4) Synchronously with the closure of the aortic clamp a solution of 2½ per cent. potassium citrate in oxygenated blood was rapidly injected until asystole occurred. The dose varied between 0.4 and 1.0 ml. of the solution per kg. of body weight. In 5 dogs the aortic clamp was released after five minutes and in 5 dogs after thirty minutes.

RESULTS

The data obtained from each study included a series of rates of cardiac inflow and the mean pressures in the left atrium and aorta recorded at each flow rate. The coronary venous return to the right atrium was not measured and constitutes an error but it is presumed that this error would be of comparable magnitude in the two series of determinations. The minute work performed at each flow rate was calculated from the formula $W = F. X \frac{(P.Ao - P.L.A.)}{100}$ where W = the minute work of the left ventricle in gramme-metres per minute, F = the minute output of the ventricle in ml. (i.e. the rate of cardiac inflow supplied by the pump) and P.Ao and P.L.A. = the mean pressures in the aorta and the left atrium respectively expressed as centimetres of water pressure.

After calculation of the minute work values for each study ventricular function curves were plotted relating minute work as the ordinate against mean left atrial pressure as the abscissa. (Fig. II). The normal curve for the left ventricle is almost a straight line and indicates that the ventricle can deal with a significantly increased load with a small rise in filling pressure. Impairment of function is expressed by a curve which is shifted to the right and has a lesser gradient. Severe impairment is expressed by curve 3 in which, at high filling pressures, the minute work falls. This is associated with cardiac failure. A rough attempt to group and quantitate changes has been made. If there is no change in function then this is signified by the letter N. If there is some shift to the right but similar levels of minute work are achieved then this effect is graded as 1. Moderate change is indicated by 2; here the atrial pressure is considerably increased and the maximum minute work is reduced. Severe impairment is grade 3; in these experiments the heart failed at flow rates which were well tolerated during the control study.

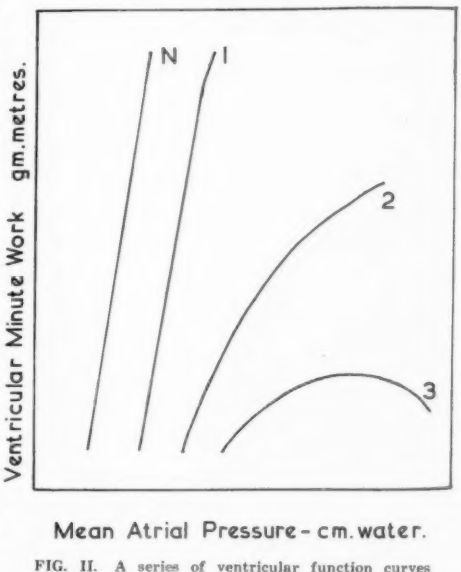

FIG. II. A series of ventricular function curves showing successive degrees of impairment of function. The control curve is on the left. If there is no change in function after insult the second curve is designated *N*. Change in function is apparent in curves graded in severity as 1, 2 and 3.

(a) *Uninterrupted aortic occlusion*

In the first group of 5 dogs the aorta was cross-clamped for five minutes. Without exception the heart continued to beat throughout this period although there was an obvious decrease in the strength and rate of contraction. The myocardium showed only slight cyanosis after four minutes and, when the

clamp was released and coronary circulation was re-established, the heart rapidly resumed a vigorous action. Left ventricular function curves constructed from data collected after a further ten minutes of cardiac by-pass showed little or no impairment of function (Fig. IV). The curves on the left of Fig. III are those of the only animal which showed any impairment of function.

The 3 studies on dogs who were subjected to ten minutes of aortic occlusion showed moderate or severe change in ventricular function in 2 and no change in one. In all biological studies one meets occasional unexpected findings and, in this series, one dog subjected to thirty minutes of aortic occlusion suffered no impairment of ventricular function.

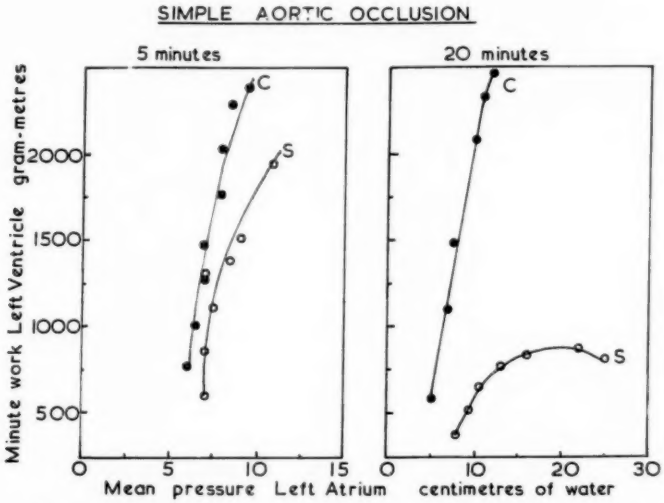

FIG. III. The results of 2 experiments. On the left the effect of five minutes of simple aortic occlusion, on the right the effect of twenty minutes of occlusion. The control curves are indicated by the letter C, the curves obtained after occlusion by the letter S. Severe impairment of function is evident after twenty minutes of occlusion.

When the aorta was cross-clamped for twenty minutes the situation was quite different. Progressive bradycardia and finally asystole occurred after six to sixteen minutes. The heart became deeply cyanosed and dilated to some extent. When the clamp was released recovery was slower and the beat, after ten minutes of further cardiac by-pass, was still noticeably less vigorous than that seen in the first group. One dog developed ventricular fibrillation which was easily reversed with a single shock. Ventricular function studies constructed from the data collected after this procedure uniformly showed moderate or severe impairment of function. In 2 dogs these studies were repeated forty minutes and sixty minutes after the release of the aortic clamp but no recovery in the functional state was apparent after these intervals.

We concluded from these studies that if the ascending aorta of a dog undergoing cardiac by-pass were occluded for a period of five minutes little if any change in ventricular function would result. If the aorta were cross-clamped for ten minutes or more significant impairment could be expected in most animals.

(b) *Interrupted aortic occlusion*

It seemed probable from the experiments analysed above that the degree of impairment of function was related to the time of uninterrupted myocardial anoxia. To test this hypothesis 5 dogs were used in a series of experiments in which the aorta was occluded for a total period of twenty minutes but after each five-minute interval the clamp was released and coronary blood flow allowed for one minute before it was re-applied. The

results individually, and as a group, were strikingly different from those obtained after twenty minutes of uninterrupted aortic occlusion. In Fig. V representative experiments from each group are compared. In 4 out of 5 experiments there was no change in ventricular function whilst in the fifth there was minimal change (Fig. VI). In fact these results are similar to those obtained after a single five-minute period of aortic occlusion.

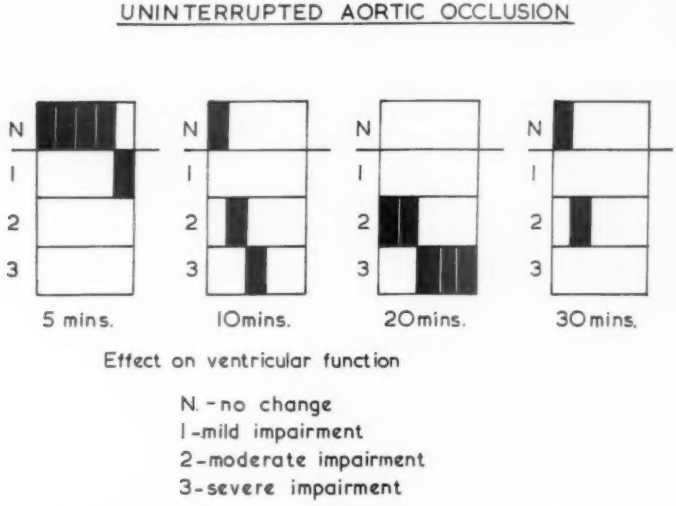

FIG. IV. The results of 15 experiments using uninterrupted aortic occlusion. The symbols N, 1, 2, 3, refer to the curves obtained after occlusion and have been graded on the basis outlined in Fig. II.

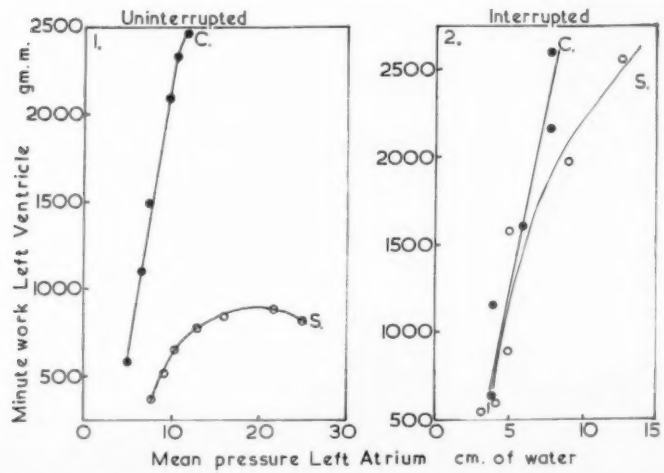

FIG. V. The effect of interrupting aortic occlusion by release of the clamp after each five minutes. There is a striking difference in these two experiments.

It was concluded from these experiments that the functional impairment which results from aortic occlusion during cardiac by-pass was related more closely to the longest period of uninterrupted aortic occlusion rather than to the total period of aortic occlusion. If the uninterrupted period did not exceed five minutes then, within limits, the effects were not additive.

remaining 2 dogs showed severe impairment of ventricular function. It may be significant that in the first 3 dogs the lowest recorded intra-cardiac temperatures were 25°C, 26°C and 28°C while in the 2 dogs which showed severe impairment the lowest temperatures were 20°C and 23.5°C respectively. One dog developed ventricular fibrillation after release of the aortic clamp but this was reversed by

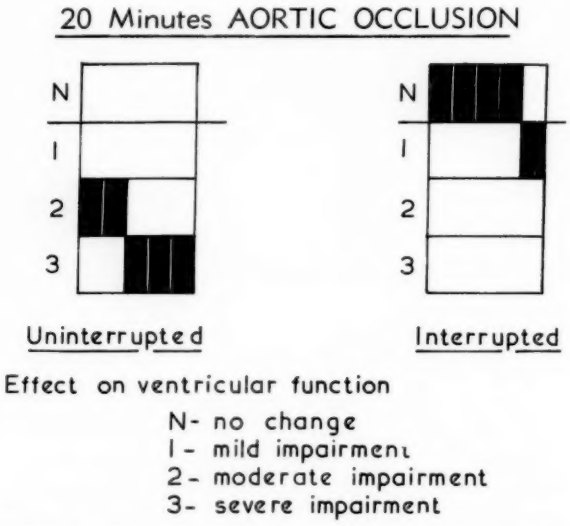

FIG. VI. The results expressed in Fig. V are seen to be typical when the 2 groups of experiments are compared.

(c) *Local cardiac hypothermia*

A series of 5 dogs were studied in which the aorta was occluded for twenty minutes and during this time the heart was bathed with cold saline so that its temperature was reduced to between 22°C and 28°C. This study was suggested by the report of Shumway, Lower and Stofer (1959) which pointed to a protective effect of local cardiac hypothermia. It was found that it was possible to drop the intra-cardiac temperature by 10°C in less than 3 minutes by this technique. When the aortic clamp was released after occlusion the heart temperature returned to 36 to 37°C in less than five minutes. It should be pointed out that the ventricular function studies carried out before and after this procedure were made when the intra-cardiac temperature was between 36 and 37.5°C. In 3 of the 5 dogs studied there was little or no detectable impairment (Fig. VII) while the electric shock. This study suggests that moderate local cardiac hypothermia may reduce the damage incurred after twenty minutes of aortic occlusion. Clearly further study is required to clarify the problem of the effects of greater degrees of hypothermia.

(d) *Potassium citrate asystole*

Ten dogs were used to study the effects of asystole induced by the technique originally proposed by Melrose, Dreyer, Bentall and Baker (1955). In each dog there was a prompt onset of asystole during injection of the potassium citrate solution. In 5 dogs the clamp was released after five minutes whilst in a second group of 5 dogs the aorta was occluded for thirty minutes (Fig. VIII). In all dogs the heart was supported by at least fifteen minutes of complete cardio-pulmonary by-pass after the release of the aortic clamp and before the second study was commenced.

Of the dogs submitted to five minutes of asystole one showed slight change, 2 showed moderate change and 2 were severely damaged by the procedure. In the dogs submitted to thirty minutes of asystole 4 showed severe change and one moderate impairment of function.

The degree of impairment of function was increased in the group which was arrested for the longer period but a study of Fig IX shows that there is a separable harmful effect of the potassium citrate injection itself.

DISCUSSION

The greatest caution should be exercised in transferring conclusions reached from the study of animal experiments, such as these, to the clinical situation in human surgery. It is quite probable that there are important differences between the responses of the dog and the human to anoxia, potassium citrate and hypothermia. The bulk of the evidence, however, suggests that these differences are quantitative rather than qualitative and it is a fact of great importance that many of the techniques now used in cardiac surgery have been developed with little modification from basic experiments using dogs. Accepting therefore that there are limitations it is possible to come to some conclusions relevant to the conduct of human cardiac surgery.

The first conclusion to be reached is that the use of potassium citrate solution to induce asystole is not only unnecessary but harmful. The advantage of this technique was that asystole was produced immediately and the profound relaxation of the heart provided ideal circumstances for repair of difficult defects. Other workers have also pointed to the harmful effects of the use of this solution. Braunwald, Waldhausen, Cornell, Bloodwell and Morrow (1959), using a similar technique, showed that simple anoxic arrest was followed by much less disturbance of function than the use of potassium citrate. After the first few minutes of myocardial anoxia, during which the heart beats to some extent, the conditions are equally suitable for precise intra-cardiac manipulation. Weirich, Jones and Burke (1959) also support this conclusion and suggest that acetyl choline is preferable to potassium citrate.

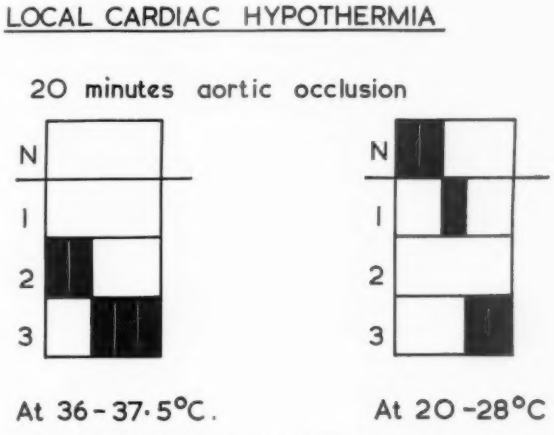

FIG. VII. Moderate local cardiac hypotherma offers some protection from damage when the aorta is occluded for twenty minutes.

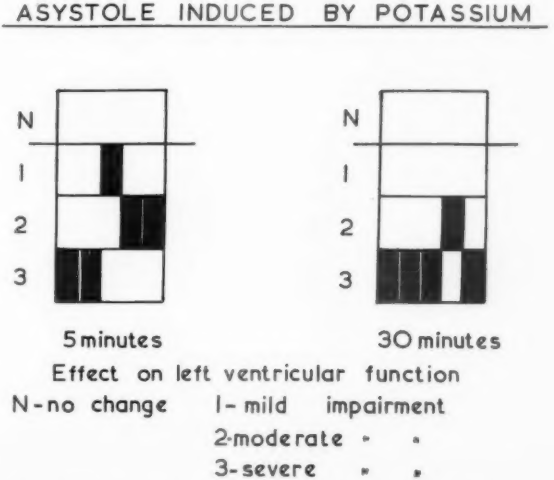

FIG. VIII. Ten experiments in which the Melrose technique was used. There was some damage in all experiments. The damage increased with increase in time of asystole.

The second conclusion that would seem to be adequately supported is that the moderately hypothermic heart is more tolerant to insult by potassium citrate or prolonged anoxia. Cooper, Willman, Zafiracopoulos and Hanlon (1959) showed that potassium asystole induced at 37°C resulted in a much greater impairment of the ventricular function than when asystole was produced at 27°C. Shumway, Lower and Stofer (1959) studied the effects of local cardiac hypothermia during anoxic arrest and concluded that hypothermia increased the tolerance of the canine heart to this procedure. Our own studies accord with this view but, in a small experience, we suspect that if the cardiac temperature is reduced below 25°C hypothermia itself may be damaging. In our experience using Drew's technique of deep hypothermia and bi-ventricular by-pass (Drew, Benazon and Keen, 1959) the right heart fails at about 25°C. This effect may be transient but there is evidence that hypothermia may produce histological changes in heart muscle which persist for some time (Unpublished data and Sarajas, Senning, Kaplan, 1956).

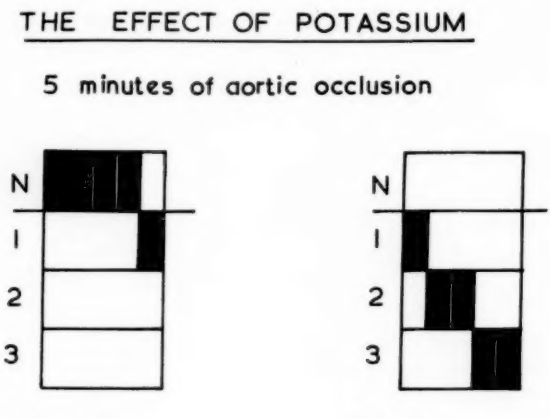

FIG. IX. A separable effect of damage by the potassium citrate is apparent when these 2 groups are compared. (a) Simple aortic occlusion for five minutes. (b) Potassium citrate asystole for five minutes.

It is clear from our own data that the impairment of function which follows total myocardial anoxia is dependent on the duration of uninterrupted anoxia. Long periods of anoxia are well tolerated if, after each five minutes, coronary blood flow is re-established for a short time. This latter technique of interrupted aortic occlusion would seem to be the most desirable for routine clinical use. If the periods of coronary blood flow are short the heart may have its nutrition rapidly restored yet not recover sufficiently in tone or strength of contraction to increase its oxygen demands to any great extent. From the surgeon's point of view it is also advantageous to keep the heart in a hypotonic state. The relative immobility and flaccidity not only allows more precise and rapid repair but it also reduces the physical trauma involved in retraction and exposure of deeply placed defects. In the direct suture repair of large ventricular septal defects with muscular edges it is important to place and tie most of the sutures with the heart in a relatively flaccid state to reduce the risk of individual sutures cutting out of the muscle during vigorous contraction. When a number of sutures are placed and tied the tension is widely distributed and this risk is lessened.

The ideal therefore for most cases is to use periods of up to five minutes of aortic occlusion interrupted by release of the aortic clamp for short periods of say one minute. If total body perfusion is conducted under

moderately hypothermic conditions it may be reasonable to use longer periods of aortic occlusion.

On occasions, particularly with large defects, surgical judgement may influence one to use longer periods of anoxia if this seems vital to the technical procedure.

It is probable that the greatly hypertrophied hearts seen in severe aortic or pulmonary stenosis have a much lower tolerance to anoxia than do those with lesser degrees of hypertrophy. In such cases even short periods of myocardial anoxia may be expected to have significant effects on function. It would seem logical in operations on aortic stenosis to use some form of direct coronary cannulation and perfusion either continuously or after every five-minute period of myocardial anoxia. In severe pulmonary stenosis with marked hypertrophy prolonged aortic occlusion should be avoided.

Whatever technique is used to produce asystole there are certain precautions to be observed. Passive distention of the myocardium invariably produces a profound depression of function. During asystole therefore the left atrium or left ventricle must be continuously decompressed and this decompression should be continued until vigorous cardiac action is re-established. In operations in which the right heart is not opened some provision for decompressing the right heart after release of the aortic clamp is essential. This can be achieved by a separate right atrial cannula or by temporary release of one of the vena-caval snares.

Our experimental experience suggests that there is a rapid recovery in ventricular function after the release of the aortic clamp. This acute recovery phase is probably completed in five to ten minutes. Slow further recovery probably occurs in hours or days. It is important in clinical practice to give the heart the maximum opportunity for full acute recovery by continuing total by-pass until this has been achieved. The impaired heart tolerates slow change in its work-load much better than rapid change of comparable magnitude. It is therefore wise to withdraw the support of the pump oxygenator slowly, during a period of partial by-pass, if there is any evidence of impaired ventricular function.

SUMMARY AND CONCLUSIONS

A study has been made of the effects on ventricular function of various methods of producing asystole.

The Melrose technique involving the use of potassium citrate invariably results in significant impairment of function. Its clinical use should be abandoned. Simple "anoxic arrest" for five minutes produces little if any change but significant change occurs after twenty minutes of anoxic arrest.

Aortic occlusion for a total period of twenty minutes but interrupted after each five minutes by release of the aortic clamp produces insignificant changes in function. This technique is advocated for clinical use.

Moderate hypothermia lessens the effects of myocardial anoxia. The significance of these observations on clinical practice is discussed.

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CONGENITAL MALFORMATIONS OF THE RECTUM AND ANUS IN FEMALE CHILDREN*

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THE complexity of the embryology of the cloaca and of the super-imposed Müllerian systems renders the rectum and anus prone to a wide range of baffling anomalies.

The anatomical variants resulting from single or combined developmental aberrations of the urinary, genital and alimentary systems seem legion.

A detailed anatomical and clinical study of many examples of these anomalies of the rectum and anus reveals that several simple external perineal patterns give the clue to their internal anatomy. Not only the internal visceral deformity, but also the associated sphincter muscles can be deduced with considerable accuracy from these external patterns. Though the anomalies are complex and numerous, they are constant in form and obey certain rules of identification.

This statement applies only to those anomalies in the anus or rectum which can be described as "communicating." Such conditions are those in which the terminal bowel communicates with the cloaca, the urinary or genital systems, or with the skin of the perineum.

Throughout this paper, descriptions apply to newborn infants though similar principles apply to older children who may have survived with the aid of bowel lavage, colostomy, or other form of remedial surgery.

The non-communicating anomalies of the rectum and anus are extremely rare in females and are always recognized in the first day or two. The malformation may be limited to the rectum and anus or it may be associated with defects in the genitalia. The internal anatomy, unlike that associated with the perforate bowel deformities, cannot be anticipated from external appearances. Recourse must be made to special radiographic techniques (Wangensteen and Rice, 1932; Stephens, 1953) which are designed to demonstrate the exact level of the occlusion. This level governs the management, the principles of which are similar to those described for imperforate conditions in the male (Stephens, 1953, 1959), anomalies which are not discussed in this paper.

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Terminology

Classification and groupings of rectal and anal deformities have been compiled by many authors including Ladd and Gross (1941), Browne (1959) and Scott, Swenson and Fisher (1960), who have included both male and female abnormalities. Many and varied terms have been used to designate the multitudinous and complex variants. There is overlap and conflict of terms which create difficulties in interpretations of writings and results.

Here I have assembled the commoner and regular deformities of the rectum and anus in the female and have evolved a scheme based on clinical anatomical and embryological data. It is heavily illustrated to clarify as much as possible this difficult series of abnormalities (Fig. 1). I have used some terms which have appeared in other writings, particularly those of Denis Browne (1951, 1959) though I have not adhered strictly to his interpretations.

This classification has proved convenient and helpful in teaching and in the diagnosis and management of the patients under my care. It is hoped that these terms and descriptions prove as satisfactory to the reader.

External Appearances

The identification of the type of pelvic deformity is difficult unless the significance and relevance of all the external features are recognized.

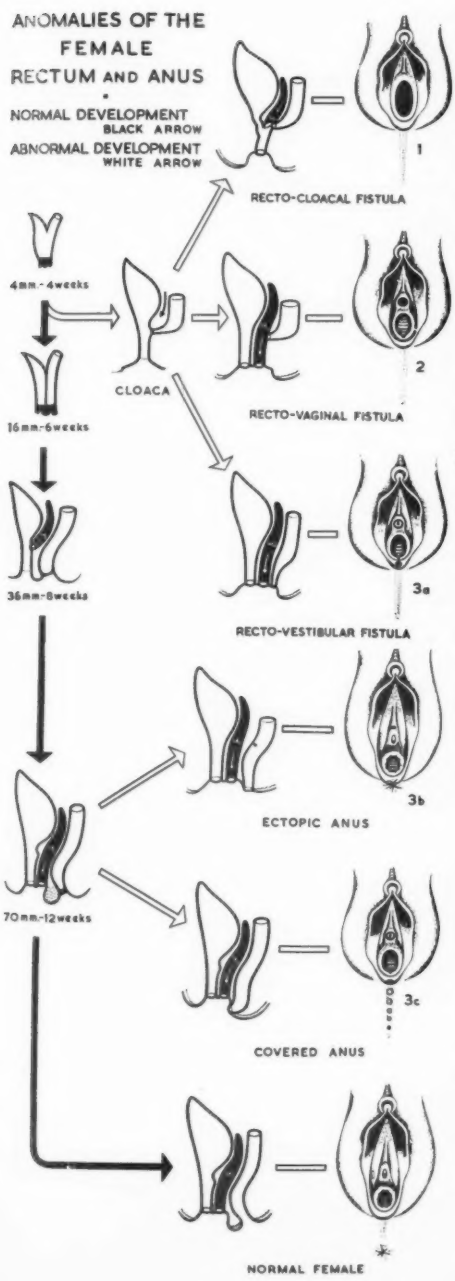

FIG. I

A special order of observation is adopted in order to ensure that none is overlooked. First, determine the patency of the terminal bowel. Secondly, identify its orifice of communication and mark out the external anatomy of those orifices which open on to the vestibule or the skin of the perineum. With these data assembled, together with the findings attending anatomical dissection (Figs. I and III), both the topography of the pelvic viscera and the arrangement of the sphincter muscles can be better appreciated.

1. Patency of bowel

At the time of birth, it may be a simple matter to tell whether the terminal bowel is perforate or imperforate. Inspection may reveal the abnormal orifice or meconium or bubbles may be visible; gas escaping under pressure may be heard especially if the baby is inverted; a probe gently steered into the likely sites may enter the rectum and produce evidence in the form of meconium. After a delay of twelve to eighteen hours, the raised intra-lumenal pressure may force the tell-tale gas or meconium into evidence. All these signs supply obvious evidence of bowel patency.

Inversion X-ray techniques may be invoked on the assumption that the bowel is imperforate, but a further intense search in the likely sites for a hidden communication is conducted in the operating theatre as a preliminary to operation.

2. Location and identification of orifices

By eye, probe or panendoscope, the rectal or anal orifice is sought and its patency established. In addition, the orifices of other organs which compromise the abnormality are charted.

Fig. I consists of three columns of diagrams — left, middle and right.

Left column: Stages in development of the normal female pelvic organs — black arrows.

Middle column: Congenital abnormalities derived from aberrations of development of the cloaca and perineum — white arrows.

Right column: Perineal patterns which depict the internal topography.

(1) Recto-cloacal fistula — 1 external orifice.
(2) Recto-vaginal fistula — 2 external orifices.
(3a) Recto-vestibular fistula — 3 external orifices all within the vestibule.
(3b) Ectopic anus — 3 external orifices — 2 within vestibule and one in skin of perineum adjacent to or contiguous with the vestibule.
(3c) Covered anus — 3 external orifices — 2 within vestibule and 1 in skin of perineum or contiguous with vestibule.

The bladder, urethra, vagina and cloacal canal can all be efficiently explored with the size 16 or 18 French sheath panendoscope using continuous irrigation to distend the lumen.

First the external orifices are examined in relation to the vestibule. Commonly, 3 regular patterns can be identified and these patterns form the guide to the internal visceral anomalies.

The perineal patterns of rectal or anal deformities in the female, fall conveniently into three groups according to the presence of one, 2 or 3 orifices respectively (Fig. I).

Group 1: The recto-cloacal fistula (Fig. I)

Only one external orifice can be found.

The external appearances of this deformity are characteristic. The vestibule is flanked by labia and the clitoris and fourchette lie in front and behind. The anterior part of the vestibule is short, shallow and moist. The single common cloacal outlet occupies the posterior half.

This single orifice is the external outlet for the cloacal canal, which is the common conduit for urine, cervical mucus and faeces and is of pencil-calibre and variable length.

Surmounting the cranial end of the common canal are anteriorly the urethra, posteriorly the rectum and the vagina in between. The lengths of urethra and vagina are inversely proportional to the length of the cloacal canal.

The urethra enters obliquely into the anterior wall. A curved sound when directed cranially and ventrally will slip into this orifice. Its calibre is normal.

The vagina opens into the vault of the cloacal canal. This organ is shorter than normal. At its junction with the cloacal canal, its orifice is partly constricted by a hymen-like structure. The cervix protrudes into its lumen at the cranial end. Sometimes the vagina is septate, in which event a sagittal septum can be identified separating the 2 orifices and 2 cervices.

The rectum enters the cloacal canal at the highest and most posterior point. Its orifice is midline and relatively stenotic.

The anal dimple is absent from the perineum which fades imperceptibly into the natal cleft.

A rarer type, already described in detail in necropsy specimens (Stephens, 1953) is the recto-cloacal fistula in which the rectum issues by a fine fistula into the base of the bladder, separating the 2 orifices of the Müllerian ducts. This rare type will not be described again in this paper but for the purposes of completeness they are included in Table 1.

TABLE 1

CLINICAL AND POST-MORTEM MATERIAL

Group	*Type*	*Patients and post-mortem material*
1	Recto-cloacal fistula	9
2	Recto-vaginal fistula	4
3	(a) Recto-vestibular fistula—	
	Common type	12
	Uncommon type	3
	(b) Covered anus	10
	(c) Ectopic anus	2

This primitive cloacal deformity is recognizable from the external appearances of the vestibule, provided that patency of the bowel is also established. The formula here is one orifice: Group 1 — recto-cloacal fistula.

Group 2: The recto-vaginal fistula (Fig. I)

Only 2 external orifices are visible; their disposition is normal but the anus is absent from the perineum. The vestibule is anatomically normal but meconium is conducted through the vagina from the rectum.

The urethra and bladder are normal in position, dimensions and function. The vagina is usually normal in size and appearance but may be septate. The hymen is well formed.

The rectal orifice is internal, lying in the midline on the posterior wall of the vagina. It is usually relatively small in calibre and lies at any level. When near the vaginal orifice it may be visible, but when more

cranially situated instrumentation is required to ascertain its location. The short rectal fistula lies in an antero-posterior axis. When traced cranially, it enlarges abruptly in diameter to that of normal rectum which, if impacted with meconium, enlarges still further.

When the vagina is partially septate, the rectal fistula issues into the single segment at a point immediately caudal to the central septum.

This pattern of 2 external orifices distinguishes Group 2—the recto-vaginal fistula.

Group 3 (Fig. 1)

Here there are 3 different types of anomaly all with 3 external orifices and resembling each other, the recto-vestibular fistula, the covered anus and the ectopic anus. I have excluded from these descriptions the uncommon true anal stenosis located at the level of the anal valves because of the lack of difficulties in diagnosis and treatment.

Recto-vestibular fistula

Here again the vestibule is normal with regard to its borders and to the urethral and vaginal orifices. It is abnormal in that it contains the rectal orifice also which is small in size, and lies totally within its borders at the site of the fossa navicularis. The rectal orifice is contiguous with or actually indents the posterior border of the vaginal orifice. Its lumen is occluded by sphincteric contraction. Retraction of the fourchette reveals a bridge of wet red epithelium of the vestibule between the rectal orifice and the skin of the perineum.

The axis of the rectum is cranio-caudal, parallel with both vagina and urethra. When a probe is introduced into the rectal orifice, it follows this cranial course; it cannot be directed posteriorly under the skin of the perineum. This probe test, differentiates the condition of recto-vestibular fistula from that of covered anus.

Close inspection and investigation reveal that there are 2 varieties of this fistula — the common and the uncommon.

The difference lies in the calibre of the distal centimetre of the fistula adjacent to the external orifice. In the common type, the expansile calibre of the rectum is normal though occluded by sphincteric compression as far as the orifice, which is usually but not always stenotic. The unusual variety exhibits a fine and delicate fistulous track leading from the anterior wall of the rectum to the external orifice. This fistulous bridge is approximately 2 cm. in length above which the rectum is of normal dimensions. The track of the fistula may follow the usual course in relation to a single vagina or it may lie in the posterior margin of the septum between the components of a double vagina. Gradings in calibre between the common and uncommon types probably occur though the author has encountered only 2 such possible examples.

Covered anus

The orifice of the anus in this deformity may lie at any point in the perineum between the normal site of the anus and the fourchette.

The genital folds are fused in an abnormal manner, in that not only do they meet in the midline anteriorly to the site of the normally placed anus but also posteriorly to this orifice, thereby creating a hypertrophied midline perineal raphe. The fused folds tent or cover in the normal anus from behind, projecting its lumen into the perineum or fourchette. The further forward the projection, the more stenotic and bevelled is the orifice.

The extremes of covering and projection, namely the most posterior and most anterior, require further description.

The anus may be only partially covered, in which event the hypertrophic raphe may be seen bridging the anal dimple in its normal location. A fine orifice may then be found on close inspection, or by probing in the depths of the dimple on one or both sides or in front of this bridge.

On the other hand, the anus may be projected to the posterior margin of the vestibule, the so-called ano-vulvar fistula, in which the appearances closely resemble those of the recto-vestibular fistula. There are 2 points of distinction. First, the bevelled orifice is contiguous with the skin of the perineum, unlike the orifice of the recto-vestibular fistula which lies totally within the vestibule. Secondly, the axis of the ano-vulvar fistula is antero-posterior. A probe when introduced

into the orifice can be passed posteriorly under the skin of the perineum. The direction taken by the probe differentiates the ano-vulvar from the recto-vestibular fistulae.

The covered anus is a common entity. Less common is the covered anus combined with covered vagina and urethra (Fig. II). The genital folds meeting in the midline project all 3 orifices anteriorly into a common canal which issues from the base of the clitoris. Furthermore, the covering in phenomenon may also be superimposed upon the patterns described in Groups 1, 2 and 3. It is then necessary to separate the folds by appropriate midline division of the covering tissues posterior to the clitoris, in order to expose the anatomy of the vestibule (Fig. II). Then the underlying abnormality can be studied and grouped according to the number of orifices exposed.

The function of this anus is perfect and no treatment is necessary. No opportunity for further macroscopic dissection or microscopic studies of the musculature has been afforded me and so no further reference will be made to this anomaly in regard to sphincteric studies or treatment.

Group 3 can be identified by the location in the vestibule or perineum of 3 separate orifices from the 3 separate systems—urinary, genital and alimentary.

MATERIAL

This work is based on both clinical and post-mortem material. Some of the early studies have already been reported (Stephens, 1953) and Table 1 represents the combined early and present series.

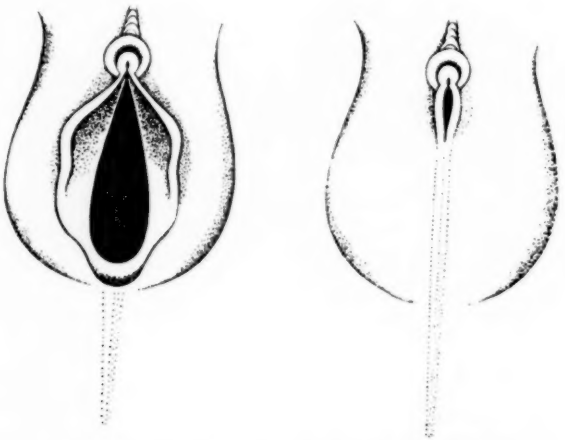

FIG. II. The usual "open" vestibule (a) and the unusual closed or "covered" vestibule (b).

The ectopic anus

In this anomaly, the anus lies anterior to the normal site. Here the perineal development is faulty. The perineum between the vestibule and the anus may be diminished or absent. The anus is sited in the dry skin of the perineum and has the normal configuration. In the event of absence of the skin bridge the anus and the vestibule are contiguous.

The rare abnormalities in the living child are the recto-cloacal and recto-vaginal fistulae. The common forms are the covered anus and the recto-vestibular fistula.

EMBRYOLOGICAL EXPLANATION OF ANOMALIES

These anomalies arise as aberrations of development at 2 distinct stages, namely, the stage of subdivision of the cloaca and the stage of perineal formation (Fig. I).

The more complex cloacal anomalies, arising at the stage when the urinary, alimentary, genital and sacral systems are actively interrelated, are frequently associated with other severe anomalies of these and other systems. On the other hand, those deformities associated with perineal formation are more often localized to the anus.

Cloacal anomalies

The cloacal abnormality caused by failure of subdivision into the urogenital and rectal compartments is at first similar for the male and female. The hindgut and urinary system remain in direct communication and their contents are conducted to the exterior through a common cloacal canal (Fig. 1).

The Müllerian systems impose female modifications upon this deformity.

The final definitive anatomical arrangement of organs depends largely on the further migratory capacity of the Müllerian bulbs. If migration is arrested the cloacal deformity persists; if it progresses the hindgut communication may be incorporated in the bulbs to form a recto-vaginal fistula or it may be advanced with the bulbs to the vestibule forming the recto-vestibular fistula.

Close study of many of these anomalies indicates that there is an infinitive number of transition forms between these 3 types, suggesting that they are all forms of the one general developmental derangement.

Perineal anomalies

Two derangements occur in the development of the perineum, hypoplasia of the perineum and abnormalities of the genital folds.

Hypoplasia leads to diminution in width of the perineum between the vestibule and the anus. The anus then lies adjacent or contiguous with the fourchette and, though normal in configuration and function, is ectopic in its location.

Fusion of the genital folds is an abnormal process in the female, though normal in the male. The bulbous and penile portion of the urethra are closed in by a postero-anterior fusion. The perineal raphe indicates this line of fusion. The common so-called adherent labia of the female may be a minor degree of fusion.

In the female, more severe grades of fusion occur. Fusion can be extensive beginning behind the anus, covering the anus and projecting its lumen to an anterior position in the perineum or to the fourchette. More rarely the fusion is even more exaggerated when not only the anus but also the vestibule are covered in, all 3 systems opening by a common orifice near the clitoris.

It is apparent that there is some discrepancy between the anatomical and embryological groupings, the reason for which is connected with the recto-vestibular anomaly.

The recto-vestibular fistula, though a cloacal deformity, is grouped into the anatomical Group 3 together with two perineal deformities, namely the ectopic and the covered anus. Not only is this malformation more easily explained as a cloacal error of development but its frequently associated sacral anomalies support its inclusion in the cloacal grouping. But for convenience of clinical classification it is better included in the anatomical Group 3.

Furthermore, the nature of development of this recto-vestibular fistula as shown in Fig. 1 and the relative frequency of the abnormalities (Tables 1 and 2) account for the scarcity of rectal deformities which lie above the puborectalis in the female. The migrating Müllerian bulbs actually carry the fistula from a level cranial to a level caudal to the levator ani musculature. The absence of this phenomenon may explain the comparative frequency of rectal deformities which lie cranial to the levator ani in the male.

SPHINCTER MUSCLES OF THE RECTUM AND ANUS

In the normal human, 3 sphincters are recognizable anatomically:

(1) the intrinsic internal sphincter, which is governed by the autonomic nervous system;

(2) the external sphincter and

(3) the puborectalis muscle, both of which are under voluntary control.

All 3 sphincters combine to provide a most efficient instrument of continence.

Important and clear cut sphincter adaptations accompany the many deformities of the rectum and anus described in this paper. Anatomical dissections and microscopic studies (Stephens, 1953) have shown that they are constant for any one type of visceral deformity. In some, the sphincters though abnormal are effective. In others the muscles, though strong, operate at a functional disadvantage and are ineffective, and in yet others the muscles are lacking or their innervation deficient.

Continence may be adequate without further surgery. On the other hand, it may be acquired only after both surgical reconstruction of the visceral deformity and correction of the functional disadvantage of the muscle. Continence cannot be expected if the muscles or their nerves are totally lacking.

It is incumbent on the surgeon, charged with the care of patients exhibiting these deformities to equip himself with precise anatomical knowledge of the involved sphincter muscles. A brief description of the skeletal muscular clothing of these deformities follows.

(a) Group 1 — Cloacal deformity (Fig. III)

The internal sphincter is totally unrecognizable. The internal and external smooth muscle coats of the rectum terminate at its junction with the cloacal canal without any thickening of the circular muscle or "coat-tail" formation (Fowler, 1957) of the longitudinal layer.

The posterior wall of the cloacal canal consists of voluntary muscle which extends proximally to the orifice of the fistula. The meeting of involuntary muscle of the rectum with the voluntary muscle of the cloaca takes place approximately at the site of the abrupt transition between rectal and cloacal epithelium on the floor of the fistula.

At the site where the normal anal pit should be in the perineum, no organized voluntary muscle can be identified though voluntary muscle fibres may be recognized lying in the sagittal plane under the skin.

The pelvic diaphragm is the muscle of major importance in the pelvis. One pubocloacal muscle sling takes the place of both the pubovaginalis and puborectalis muscles. This muscle is closely and firmly attached to the posterior and side walls of the cloacal and to the caudal aspect of the recto-cloacal fistula. This U-sling operates as a cloacal compressor, though at a functional disadvantage upon the rectal orifice, so that it is an inadequate rectal sphincter.

The pelvic diaphragm stretches from its lateral bony origins to a midline raphe which extends from the pubocloacal muscle anteriorly to the coccyx and sacrum posteriorly. Its configuration and action closely resemble the normal muscle. It is an effective pelvic support and diaphragm.

(b) Group 2 — Recto-vaginal fistula (Fig. III)

Here expansions of the intrinsic muscle of the rectum at the junction with the vaginal wall are unrecognizable microscopically or macroscopically. The 2 layers of smooth muscle end abruptly without thickening at the junction of the fistula with vaginal mucosa on the vaginal wall. The smooth muscle layers of the vagina overlap the rectal muscle for a short distance proximal to the mucosal transition.

No recognizable external sphincter muscle is formed around the normal site of the anus but, again, longitudinal striped muscle fibres course through the fat in an antero-posterior direction.

The levator ani diaphragm resembles closely that of the cloacal deformity.

The pubourethral and pubovaginal muscles replace the pubocloacal muscle. When the rectal fistula is high on the posterior wall it is cranial to this muscle sling; when low and the sling obtains a direct action on the caudal lip of the orifice of the fistula, the sphincteric action of this sling of muscle is more effective.

(c) Group 3 — Recto-vestibular fistula, covered anus and ectopic anus (Fig. III)

Here the orifice of the alimentary system is external and the anal canal lies within the pubovisceral sling. The rectum is potentially continent but local anoplasty is usually needed to enlarge the orifice or to correct its axis.

Recto-vestibular fistula

Only one complete specimen of the common type of recto-vestibular fistula was available for microscopy studies of the sphincter muscles.

The intrinsic muscle layers of the bowel showed slight thickening, suggesting the presence of an internal sphincter.

The pubovisceral sling of the levator ani muscle hugged the back and side walls of the rectum near its orifice. Caudal to and blending with the lowermost fibres of this U-shaped puborectalis was a narrow collar of voluntary muscle, which embraced the orifices of the urethra, vagina and rectum.

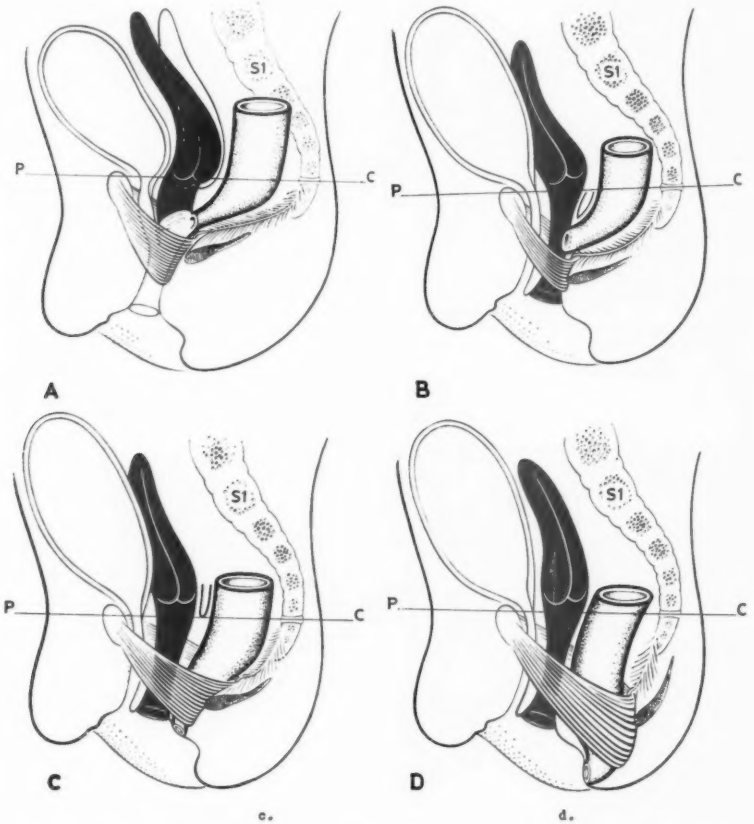

FIG. III. Four diagrams to show the pubovisceral sling and diaphragm of the levatores ani musculature in the recto-cloacal fistula (a), recto-vaginal fistula (b), recto-vestibular fistula (c), and covered anus (d). Figs. IIIa, b and c are drawn from anatomical dissections and Fig. 3d is composed from clinical assessment only. P-C indicates the pubococcygeal line.

Again, the voluntary muscle fibres representing the rudimentary external sphincter were arranged in the sagittal plane in the fat beneath the skin of the perineum.

The pelvic diaphragm was a strong sheet of voluntary muscle.

The action of the U-shaped sling can be detected by digital examination of this orifice in older children.

Patients exhibiting this deformity, though treated by simple dilatation only, usually exhibit satisfactorily effective rectal continence.

Covered anus

No specimen of this anomaly in the female has become available for dissection or microdissection. From operative experiences, digital examinations and clinical estimations subsequent to cut-back procedures (Denis Browne, 1951), the following deductions have been made:

(a) The internal sphincter is deficient; the "anus" after operation gapes, exposing the red mucosa of the rectum.

(b) The external sphincter in its superficial part is also judged to be deficient for the same reason. The deep part of the sphincter seems to be present because, in the co-operative child, a definite circumferential squeeze adjacent to the outlet of the rectum is palpable during digital examination. Not only is the compression detected on the posterior and lateral aspects but also anteriorly, and it is presumably caused by the encircling musculature of the deep part of the external sphincter.

(c) The puborectalis sling can be palpated digitally, extending over several centimetres of rectal wall above the outlet. In a co-operative child, the compression is effective posteriorly and to a lesser extent laterally, but not anteriorly at this higher level.

The net result of this adaptation of sphincters is a high grade of voluntary control.

The ectopic anus

This is an unusual anomaly in which the anus appears normal except for its anterior situation. No opportunity has been available for detailed anatomical or functional studies. Normal voluntary control can be expected.

Practical Considerations in Regard to Rectal Continence

The following practical considerations are relevant when planning the management of each type:

1. The orifice of the rectum lies cranial to the pubovisceral sling in Groups 1 and 2 and caudal in Group 3.
2. The pubovisceral sling acts chiefly on the common outlet (cloaca or vagina) and at a considerable disadvantage on the rectal orifices in Groups 1 and 2, but in Group 3 it acts directly and effectively on the rectum.
3. In order to enhance the effect of the pubovisceral sling in Groups 1 and 2, operative procedures are necessary to redirect the rectum to the exterior so that the anal canal becomes embraced by the U-shaped band or pubovisceral sling.
4. The puborectalis sling is sited correctly for good functional efficiency in Group 3.
5. The internal sphincter is rudimentary in all 3 groups. The pubovisceral sling, though modified, is the chief effective sphincter muscle in all and in Group 3 the deep part of the external sphincter may contribute to sphincteric efficiency.

Neural Impairment of Sphincters

When the sacrum is radiographically complete or lacks only its fifth segment, the nerves to the sphincters and levator ani musculature may be presumed to be present.

Varying degrees of sacral agenesis are not uncommon in association with cloacal deformities, namely Groups 1, 2 and 3a (Fig. 1), because at the time of cloacal subdivision, the terminal spine is in the process of formation.

A rough guide only to the nerve supply to the actual or potential sphincter mechanisms can be obtained from radiographic assessment of the sacral vertebrae. Here the phenomena of epistasis (Williams and Nixon) and hemi-vertebrae may confuse the vertebral count.

Smith (1959), having dissected the sacral nerves in 6 specimens of sacral agenesis extending to various levels, found that in 4 the neurological and vertebral deficiencies corresponded exactly but that in 2 the anatomical nerve segments extended beyond the level of the vertebral deficiencies. This neurological "overflow" explains the occasional clinical observation of perineal sensory appreciation when the agenesis involves the third, fourth and fifth sacral vertebrae (Williams and Nixon, 1957; Smith, 1959). Furthermore Smith (personal communication) is disinclined to assess potential sphincter function

of the associated rectal deformity on radiographic evidence alone in agenesis of the sacrum. In the final prediction prior to surgical correction of the rectal deformity, the analysis of the readily assessable sphincters and detrusor of the urethra and bladder, and of perineal sensation provides the important key to the puzzle. Normal bladder and urethral function foretells a potential puborectalis muscle. Absence of perineal sensation indicates absence of sphincter competence of both bladder and bowel but, conversely, the presence of perineal sensation alone does not provide reliable evidence of sphincter efficiency of either system.

When the rectal or anal deformity co-exists with a myelomeningocele, the assessment is made on similar clinical and radiographic studies. The upper limit of the spinal deformity can usually be fairly accurately assessed and the vertebral arches caudal to this level are presumed to be defective. The spinal cord is dysplastic and impaired in function throughout the zone of spinal deformity and in some patients for a varying distance cranial to it.

In the radiographic assessment of neural damage therefore, it should be borne in mind that a neural "overflow" may exist beyond the level of the most caudal vertebra in sacral agenesis and that the neural outflow may be impaired proximal to the spinal deformity in myelomeningocele.

On the evidence derived from the above investigations a decision should now be made to proceed with or abandon plans for reconstruction of the anal canal.

TREATMENT

Having studied and correlated the external and internal anatomy and having assessed the state of the nerve supply to the levator ani and external sphincter muscles, the surgeon may then decide on the plan of reconstruction. He is faced with 3 alternatives; to transpose the orifice of the anus from the abnormal site to the normal site, to dilate or enlarge it in its abnormal site, or to resort to colostomy. For this anomaly in its multitude of variants, any one of these may be applicable. Suffice it to say at this point that the final form of the anus and anal canal should have the following qualities:

(1) it should open externally as in Group 3 not internally as in Groups 1 and 2;

(2) it should be directed caudally as with Groups 3a and 3b and not ventrally as in Groups 1, 2 and 3c (Fig. I);

(3) it should be supple and adequate in size, and

(4) it should lie within the grasp of the pubovisceral sling of the levator musculature as in Group 3 and not above the sling as in Groups 1 and 2.

In devising specific plans for the treatment of the several anomalies the foregoing criteria should be kept in mind.

It is apparent that to achieve these objectives a rectoplasty is necessary for the cloacal and recto-vaginal anomalies, whilst an anoplasty will fulfil the needs of the remainder.

Rectoplasty

Surgeons differ in their opinions regarding the use of preliminary defunctioning colostomy. Some prefer the one stage immediate reconstruction (Rhoads *et alii*, 1948); others prefer the preliminary colostomy procedure. There are some advantages in staging the operation, for example, definitive surgery is carried out in a clean field, the major procedure can be delayed a year or more until the pelvic dimensions have enlarged and healing of the muco-cutaneous suture line takes place in favourable circumstances. A transverse colostomy in the author's view is a prerequisite of the rectoplasty, even though it introduces multiple operations with their small attendant risks.

Again, surgeons differ on the surgical approach to the definitive procedure. Many favour a combined abdomino-perineal reconstruction (Rhoads *et alii*, 1948; Santulli, 1952; Potts, 1959). In this method, the detachment of the fistula and the tunnelling of the pelvic musculature are carried out from the abdominal aspect. The delicate and crucial phase of the operation, the enlargement of the puborectalis sling, is performed in the depths of the wound under difficult circumstances.

The author, for reasons largely based on the appearances of the dissected specimen, prefers to approach the puborectalis sling by

the more direct sacro-coccygeal route. Here the surgeon can clear the convex caudal wall of the rectum and the fistula under vision in the supra-levator space; he can divide the fistula and then direct the blades of a right-angled forceps carefully and accurately parallel and posterior to the cloacal or vaginal wall to the skin of the perineum, through the sling of the puborectalis muscle and through the tough tissue of the perineal body. The special features observed on dissection which justify this approach are the intimacy with which the sling blends with the posterior wall of the cloaca and the caudal wall of the fistula and the delicacy of this sling. By this approach the tunnelling of this musculature can be accurately executed with minimal trauma, even though the diaphragm and sling are not readily visible.

cision through the junction is made and a small window is opened into the supra-levator space.

The thick fascia covering the terminal portion of the bowel is incised transversely across the long axis. Once the plane of the rectal wall itself has been reached, the tissues can be freely separated to expose the fistula down to the vaginal wall. The rectum close to the fistula is divided. The severed end of the bowel is lifted through the incision, oversewn and further mobilized by separating the bowel wall from the surrounding structures in the same plane.

The severed distal end of the fistula lying deep in the incision is oversewn when possible.

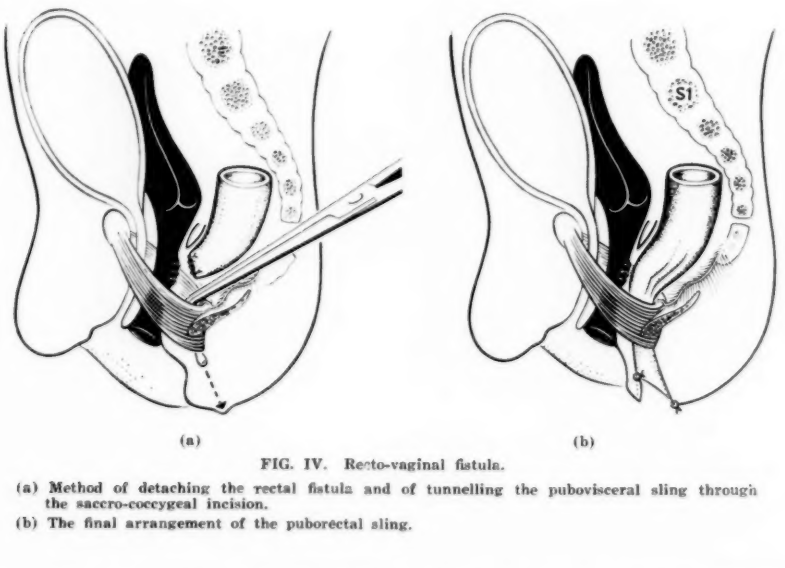

FIG. IV. Recto-vaginal fistula.

(a) Method of detaching the rectal fistula and of tunnelling the pubovisceral sling through the saccro-coccygeal incision.

(b) The final arrangement of the puborectal sling.

The procedure is designed to provide the new anal canal with a sphincter composed of the carefully preserved pubovisceral sling.

The sacro-coccygeal approach (Stephens, 1953) has been developed for the detachment of the rectal fistula and the accurate tunnelling of the pubovaginal sling (Fig. IV).

A midline natal cleft incision is made through the skin with its centre over the sacro-coccygeal junction. A transverse in-

The second important step in the operation concerns the pubovisceral sling. Neither this sling nor the levator diaphragm is well visualized through this approach. But on the assumption that the muscle is present, the tunnelling can be accurately performed. The tunnel, only large enough to permit the passage of the new anal canal, should lie contiguous with the posterior wall of the vagina, below the fistula, in the mid-sagittal line.

The passage is made by directing the blunt ends of a right-angled forceps parallel and contiguous with the posterior wall of the vagina. This manoeuvre is facilitated if the vaginal wall is steadied by a vaginal obturator, either a Hegar's dilator or, in older children, by the index finger. The tips of the forceps are advanced caudally to the level of the vestibule and turned posteriorly under the skin of the perineum to the site of the new anus. There a perineal orifice of the "trapdoor" pattern is made.

The trapdoor skin incision is designed as 3 sides of a square, the fourth uncut side forming a flap hinged anteriorly. This flap is later inturned and integrated into the anal canal. The tape is drawn through the tunnel from perineum to pelvis and out through the sacro-coccygeal incision. It is tied loosely externally. This tape serves 2 functions. It is a temporary convenient guide to the tunnel and it can be used to stretch the lumen.

The third step is the mobilization of the rectum. This is done by blunt dissection of pararectal tissues from the rectal walls on both sides.

At this stage of the operation, the surgeon must decide whether the rectum can be mobilized sufficiently to enable the whole operation to be completed from the sacro-coccygeal route or whether it is necessary to proceed to an abdominal operation to gain the extra mobilization.

If mobilization is adequate, the rectum is squeezed through the tunnel and sutured to the skin of the perineum (Fig. IV). The flap of perineal skin is dove-tailed as a gusset into the anterior rectal wall.

The trapdoor incision has 4 merits—

1. It enlarges the anal orifice;
2. the long anastomotic line inhibits stricture;
3. the anterior hinged wall ensures a wider skin bridge between the vagina and anus;
4. if the length of mobilized bowel is insufficient anteriorly, the gusset fills the deficiency.

For the low-lying rectal fistula, rectoplasty procedures can be completed by the sacro-coccygeal route. For the high fistula when greater length of rectum is necessary to fashion the new anal canal, an abdominal approach is also required. The sacral incision is then sutured, a short glove drain is inserted and the child is then turned into the lithotomy position.

The pelvis is explored from the abdomen. The rectum is freed from the pelvis by liberal division of the peritoneum in the pelvic floor. The sigmoid colon with its blood supply is mobilized to give that extra length required for the rectoplasty. Sometimes the distal part of the sigmoid is abnormally large in calibre in which event resection of this part is advisable.

Turning now to the perineum, the operator exerts traction on the tape to open up the tunnel, the rectum is eased through and anastomosed to the trapdoor incision of the skin. The tape is now removed.

The pelvic peritoneal floor is then reconstituted and the abdominal incision is finally sutured in layers.

After the immediate post-operative phase, gradual digital dilatation of the puborectal sling through the newly constructed anal canal can be carried out by the surgeon or by the mother.

As a separate stage, the colostomy is closed at a convenient time.

Anoplasty

On recognition of the Group 3 abnormality in the newly born infant, immediate remedial surgery should be instituted before the rectum becomes over-distended with meconium or faeces. Here there is no need for a defunctioning colostomy in the majority of patients.

The orifice of the common form of recto-vestibular fistula is usually stenotic. This requires dilatation, sometimes assisted by incision of the tough posterior margin, which is otherwise resistant to simple dilatation procedures. Furthermore, unless this margin is divided, dilatation may rupture the thin septum situated anteriorly between the vagina and rectum.

The uncommon variant of this abnormality with a fine delicate elongated rectal fistula opening to the exterior at the posterior margin of the septum between the components

of a double vagina in the vestibule, is not so amendable to dilatation. The passage of sounds along the fistula may rupture the adjoining vaginal wall, converting the malformation into a low recto-vaginal anomaly. Instead of a simple anoplasty, a rectoplasty is necessary.

The orifice of the covered anus not only lies in an anterior position but is directed anteriorly. Furthermore it is stenotic.

RESULTS OF TREATMENT

Following rectoplasty the level of action of the only sphincter muscle, the puborectalis, lies approximately 5 centimetres internal to the muco-cutaneous junction. Its action is strong, effective and easily palpable on rectal examination of the co-operative child. The anal canal is continent for formed faeces by day and by night from the first few weeks after this form of rectoplasty though soiling

TABLE 2

RESULTS OF RECTOPLASTY AND ANOPLASTY OPERATIONS ON CONTINENCE

	Rectoplasty	*Anoplasty*	*Puborectalis sphincter muscle (Digital assessment)*	*Results*	*Remarks*
Recto-cloacal fistula	1		U-sling weak. Situated high in pelvis	After 3 years has poor voluntary control; still improving	Anus moistened with mucus. Was mentally retarded but now improving
Recto-vaginal fistula	2		U-sling strong and situated high in pelvis	Good continence for formed stools	Anus moistened with mucus
Recto-vestibular fistula					
Usual type	1		U-sling strong and high in pelvis	Good continence even with fluid stools	Faecal smear unless special toilet care after defaecation
		6	U-sling strong and low in pelvis	Excellent continence	Clean. No mucus leakage
Unusual type	3		U-sling strong Situated high in pelvis	1—excellent continence 1—good continence except for diarrhoea stools (dietetic indiscretion) 1—death—early post-operative haemorrhage	Need special toilet care after defaecation
Covered anus		7	U-sling strong and low in pelvis Circumferential squeeze at orifice palpable	Excellent continence	Clean. Anus slightly moistened by mucus
Ectopic anus	—	—		Good continence. No operation needed	

Treatment is simple (Browne, 1951). A midline incision, extending posteriorly from the orifice as far as the normal site of the anus, is made in the skin. The incision divides the rim of the orifice and lays open the skin and bowel lumen. The calibre of the rectal orifice should be large enough to admit the tip of the fifth finger.

Suturing of mucosa to skin is immaterial. Retraction of the bowel does not occur; healing is rapid and scar formation is most unusual. Dilatation with the fifth finger tip on alternate days by surgeon or parent for the first few weeks should be performed. This prevents re-fusion of the genital folds.

accidents may occur with bouts of diarrhoea or indiscretions of diet. Furthermore, mucus secreted from this sleeve distal to this sphincter moistens the peri-anal skin. Toilet ingenuity on the part of the mother and the child are necessary to ensure cleanliness.

Similar findings have been observed in a larger but analogous series of rectoplasty procedures in male patients.

Following anoplasty, the main muscle of continence is again the puborectalis muscle, but here the effective level of action of this muscle extends to within several millimetres of the muco-cutaneous junction. If this muscle is reinforced by the deep part of the

external sphincters as in the covered anus the distal sleeve is short but, even so, the gaping external orifice at the muco-cutaneous junction allows slight mucous wetting of the perineum. Continence in this group is highly efficient.

Special mention should be made of the common form of recto-vestibular fistula. Here the puborectalis extends down to and envelopes the orifice in the vestibule. The orifice is snug, shut and inconspicuous. Its sphincter mechanism is highly effective against faeces, flatus and mucus and can withstand bouts of diarrhoea. Moreover, the anus, vagina and vestibule are unsoiled. This anomaly carries with it the minimum of inconvenience and is compatible with an entirely normal social existence.

Not all persistent faecal soiling is caused by organic lesions of the nerves or sphincters. Constipation with overflow may afflict a normal or an abnormal child.

Soiling by overflow in a constipated, otherwise normal, child is a common entity. Accumulations of firm faeces become impacted in the rectum, partially blocking the lumen. The rectum becomes an over-distended reservoir, insensitive and unable to evacuate its bulky scybala. Newly formed soft faeces are massaged by peristalsis to the exterior past the scybala as a continuous night and day faecal discharge.

This same syndrome may be superimposed upon either the rectoplasty or anoplasty operation as described for the several anomalies which form the basis of this paper. Stenosis before or after operation causes rectal dilatation which in its own right is a precursor of faecal overloading of the rectum. Not only must the stenosis be adequately managed but also the rectal dilatation. Careful post-operative attention will prevent the syndrome but once established it is of the utmost importance to differentiate between faecal soiling of the overloaded rectum and true incontinence. Spurious incontinence can be simply alleviated but true incontinence calls for radical measures.

One or more thorough courses of conservative management including total evacuation of faeces, bowel washouts (not enemas) over a period of several weeks or months, bowel stimulants and training, may be necessary to ensure success (Stephens, 1956).

A thorough trial of conservative management is essential not only for treatment but for assessment of sphincteric competence.

From this account, it is obvious that the imperfections of anatomy after surgical repair of these congenital anomalies impose corresponding minor or major impairments of control of faeces and mucus. If the action of the puborectalis muscle is preserved adequate faecal control is possible, though extra toilet care is necessary to maintain proper cleanliness.

SUMMARY

The communicating anomalies of the rectum and anus in the female have been grouped according to the external anatomical patterns:

Group 1, one orifice—recto-cloacal fistula.

Group 2, two orifices — recto-vaginal fistula.

Group 3, three orifices — recto-vestibular fistula, ectopic anus and covered anus.

This grouping is designed to commit indelibly to memory a simple classification in which the number of the group is identical with the number of external orifices. In each group, the particular anomaly is tagged by a descriptive term which is perhaps more expedient in the vocabulary of clinical surgery. They connote regular patterns upon which the surgeon may presume the general internal visceral anatomy.

When the sacrum is normally developed, the levatores ani musculature is present but important and regular adaptations accompany the various deformities.

When estimating the degree of neural disturbance of the sphincters, especially in the concealed rectal anomalies in Groups 1 and 2, the greatest help comes from the clinical observation of the urethral control. If the neural control of micturition is efficient, then it can be presumed that the various adaptations of the levatores ani musculature are also actually or potentially efficient.

Of all the sphincters of the rectum and anus in these anomalies, the U-sling of the levatores ani musculature is the chief instrument of continence and is indeed the key muscle in successful operative reconstructions.

The deformities in Group 1 recto-cloacal fistula, Group 2 recto-vaginal fistula, and Group 3a recto-vestibular fistula, are examples of aberrations of cloacal subdivision, whilst Group 3b ectopic anus and 3c covered anus, are derived from defects of formation of the perineum.

The surgical treatment of abnormalities of Groups 1 and 2, consists of a preliminary defunctioning colostomy, followed by reconstruction of the anal canal — a rectoplasty; for Groups 3a and 3c, neither colostomy nor rectoplasty is required and anoplasty is adequate for the purpose.

Provided that the neural outflow to the sphincters is unimpaired and provided that the U-sling of the levatores ani is carefully preserved, a high degree of continence is possible after surgical reconstruction of the anal canal.

Acknowledgements

In my earlier articles on this subject (1953), I made sincere acknowledgements to the medical staffs of both the Hospital for Sick Children, Great Ormond Street, London, and the Royal Children's Hospital, Melbourne. Again, I owe thanks to both institutions even though ten years have elapsed for the principles which were developed then still apply. The Nuffield Foundation has helped financially with the cost of the illustrations, which were drawn for me by Mr. Eric Thake. Dr. Reginald Webster has again assisted me with advice on the preparation of this paper and in the correction of the manuscript. I am duly grateful for this kindness.

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THE BY-PASS OPERATION AS APPLIED TO THE LEGS*

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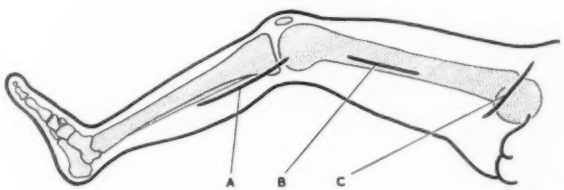
THE introduction of the by-pass operation by Oudet was dependent on 2 factors. The first was the realization that atherosclerosis, although a biochemically diffuse disease, often remains segmental and localized in its clinical manifestations for many years. Secondly, there was the advent of suitable tubes for the operation, originally human arteries or veins, more recently the synthetic materials, of which crimped Teflon is the best. In the background was the knowledge of the geometry of arterial disease derived from the intensive arteriographic studies in previous years and the familiarity with human arterial techniques initiated by recent experience with direct arterial replacement. Like many other procedures this operation has been overexploited, often with damage to the patient and it may be salutary to review the present trends regarding its indications, technique and outcome.

The indications we have rigorously defined as (a) unilateral disease, which is an economic hazard to the patient and (b) the salvaging of a limb threatened by minor or major gangrene. It is immediately evident that of any group of patients coming to a vascular clinic with the complaint of intermittent claudication few will be selected for the operation. Patients are mainly excluded on the grounds of bilateral disease or because in those suffering from predominantly single-leg disease the arteriographic studies show occlusion or gross involvement of the popliteal segment. Indeed, probably not more than 2 or 3 per cent. of claudications are suitable for the by-pass. Some other factors must be considered; age and associated cardiac or cerebral episodes are among these. Although anaesthesia and resuscitative measures have brought the surgery of the aged to high standards of safety, we are unwilling to operate on patients over say 65 for purely social reasons. One cannot accept, for example, the inability to walk round a golf course as a valid reason for surgical intervention. Also, as time goes on we are more wary of the patient who has, in addition to femoro-popliteal disease, additional stenosis or further discrete occlusions in the tibial vessels. Such arteries will not accept the "run-off" from an inserted graft and it is common experience that such cases do badly. Another difficulty we have run into is with arteriograms which, though obligatory before grafting, provide difficulty in the interpretation of the popliteal segment. In single plane films one is sometimes deluded in believing the artery is unencroached upon by atheromatous plaques, whereas exploration uncovers a vessel half occluded by clot and unfit for grafting. Where a disobliteration of the popliteal segment has been necessary to prepare the artery for grafting, our attempts have been consistently unsuccessful.



LOWER LEG, UPPER & LOWER THIGH INCISIONS

FIG. I

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TECHNIQUE

The technique of the by-pass operation, as originally described, consisted of running a graft, inserted in the superficial femoral artery, through Hunter's canal into the popliteal fossa, at which stage the patient was turned onto his face and the lower anastomosis completed in the prone position. Rob improved on this by operating with the patient lying on his side with the affected leg uppermost, extended and abducted. By this means a synchronous combined operation could be performed with one operator dissecting the groin and one the popliteal fossa. This we have found cumbersome because it makes the groin difficult to approach. These positions have, therefore, been abandoned and we have developed over the past two years a technique (which has recently been described by Morris *et alii* (1959) and can be performed with the patient lying supine. It has the additional advantage of exposing the popliteal artery at its lowermost end, where experience has shown that it is likely to be least involved in disease and, because of the ease of access, allows one operator comfortably to complete both anastomoses. The technique is illustrated in Figs. I to IV. We first expose the popliteal artery to ensure that it is suitable for grafting. The incision of about 15 cm. is made behind the medial border of the tibia staying in front of the internal saphenous vein and nerve. Immediately below the skin and in the same plane the tough fascia covering the medial belly of

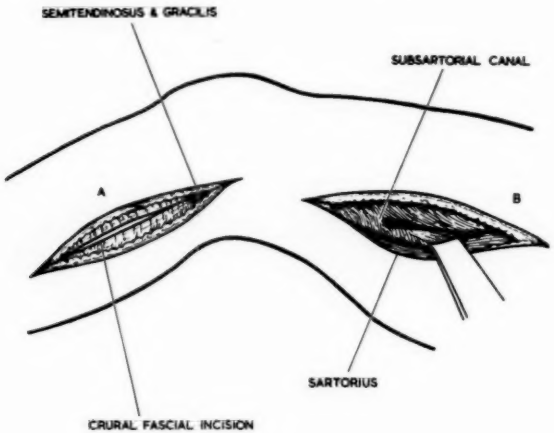

FIG. II

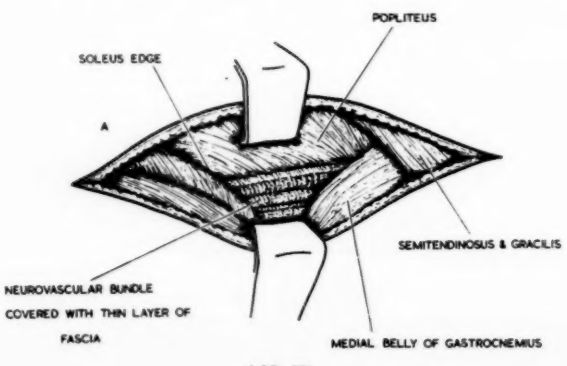

FIG. III

gastrocnemius is incised and the muscle itself exposed. This is very easily mobilized forwards by finger or blunt dissection so that the popliteal neuro-vascular bundle is exposed in its thin connective tissue sheath. The bundle is traced below to where it disappears below the soleal arch and above can be traced to almost the level of the knee joint. If necessary the semitendinosus tendon can be partly divided to increase the height of exposure. The artery is then dissected out from its accompanying structures and elevated towards the surface of the incision by 2 nylon tapes. The length the popliteal artery runs below the knee joint before it divides differs considerably and has ranged in the last 30 cases between 2.5 and 6.5 cm. Occasionally the bifurcation may occur above the knee

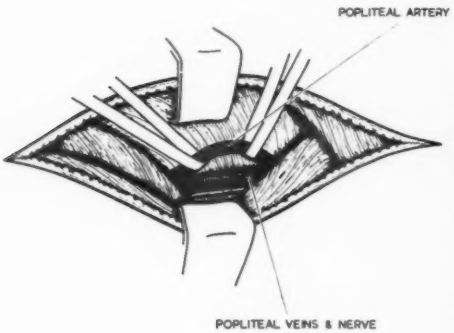

FIG. IV

joint and, of course, the anterior tibial artery may occasionally follow its embryological path and proceed to the anterior compartment in front of the popliteus muscle. Such exceptions will be picked out by the preoperative angiogram and excluded from such a technique. A glance at anatomical textbooks leads to the impression that the lower or middle genicular tributary may arise from and interfere with the dissection of the popliteal segment. In practice this is not so as these vessels consistently arise above the level of the knee joint.

Provided the popliteal artery is of such size and condition to be graftable, the common femoral artery is next exposed in the groin, a disc of its anterior wall is excised and the proximal end of the graft is inserted by a running over-and-over 5/0 silk suture. If a synthetic is being used temporary release of the proximal clamp used for controlling the femoral artery will flood the graft whose walls are thus preclotted. A third short incision is made over the lower end of Hunter's canal to expose the femoro-popliteal segment at the level of the adductor opening. With finger dissection the lower intermediate and upper incisions can be connected. It is important to see that the graft runs smoothly around the adductor opening which may on occasions need enlarging by dividing part of the adductor magnus insertion. The graft is

FIG. V. Pre-operative arteriogram.

now washed out with saline to exclude any reformed clots and worked, without twisting or torsion, down the prepared track. The popliteal artery is now temporarily occluded, its wall incised, an ovoid piece excised and the lower end of the graft inserted. The clamps are temporarily released before the final few sutures are put in. The incisions are closed without drainage. Anticoagulation is started four days post-operatively, if this is thought to be indicated. The peripheral pulses may be immediately palpable after the operation or their return may be delayed up to three days.

DISCUSSION

In the successful cases which are a function of technical skill and accurate selection of cases, the results are striking. The lame walk without handicap, the maimed heal. The following case is illustrative. A male patient, aged 57, whose occupation as a bus driver was threatened because the use of the left leg on the bus pedals caused calf claudication. He was generally fit and there was no evidence of gross disease except in the left leg where an arteriogram (Fig. V) revealed a 20 cm. thrombosis of the femoral artery. The popliteal segment, although invaded by atherosclerosis, was of reasonable diameter. A crimped tube was inserted from the common femoral to the lower popliteal segment (Figs. VI and VII). His pulses remain palpable one year after the operation and the patient has returned unhandicapped to his previous occupation.

FIG. VI. Arterial flow through crimped dacron tube.

FIG. VII. Lower end of dacron tube showing entrance in lower popliteal artery.

Even the successful cases are, however, a calculated gamble for it is not infrequent for the disease process in the months following operation to spread proximally or distally in the involved artery to cut off the anastomotic line, or a delayed thrombosis occurs in the graft itself. Even more important is that, with very careful prior selection, the immediate failure rate (that is within twenty-four hours) of by-pass grafting in the legs is, in my hands, about 50 per cent. In the majority the failure is attributable to clotting on the graft side of the lower anastomosis. This thrombosis rate seems partly dependent on the type of graft inserted and we strongly believe that from an immediate and long-term point of view vein grafts are superior to the synthetics. Their dissection unfortunately adds an hour to the operation and a suitable length or diameter of vein is not always available. As the commonest site for failure is at the popliteal anastomosis efforts have been made to predict the feasibility of successful grafting by direct pressure readings in the segment at the time of operation and by measuring the vigour of the flow-back from the incised artery distal to a temporary clamp. Neither of these criteria has proved much of a guide. Nor can we claim that a failed by-pass leaves

the limb no worse off than before operation. Two patients in whom a crimped Dacron graft was inserted developed massive deep thrombo-phlebitis at one week and two months respectively. In one diabetic patient the graft had to be removed due to a low grade sepsis developing at the proximal suture line. In several cases the seizing-up of the graft was associated with an exacerbation of the foot ischaemia, so much so that our present practice is to perform a lumbar sympathectomy at the time of the by-pass procedure.

Our experience would, therefore, suggest that (a) the chief indications for a by-pass operation in the legs are limited to a small percentage of atherosclerotic claudicants who are economically handicapped, or whose limbs are nutritionally threatened. It may well be that the important future of the by-pass will be in the treatment of major arterial lesions associated with acute trauma; (b) although the successful cases are impressive there is a high immediate and delayed failure rate such as one might predict with such a widespread and progressive disease state; (c) the operation is not without morbidity and should not be lightly undertaken.

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THE RESPONSE OF THE ENDOCRINE SYSTEM TO SURGERY*

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TO attempt in a short paper a general review of this vast subject is to essay an impossible task. All I can do is to paint in very broad strokes, and as well as I can, a picture of modern views about it. I will exclude from consideration the effect of surgery directed at any of the endocrine organs themselves, either for disease of that organ (such as hyperthyroidism, phaeochromocytoma, functioning pituitary or thyroid adenoma, and the like) or as a therapeutic measure. This leaves in effect the role of the endocrine organs in the general metabolic response to the trauma of surgery.

Cuthbertson (1930, 1934) was one of the first to interest himself in this subject, mainly from the nutritional aspect. His work, and other early studies, are summarized in a review published in 1945. During the second World War there was a good deal of work on surgical endocrinology such as that of Albright (1943) and Browne *et alii* (1944), while in 1946 Selye published his concept of the "alarm reaction" as a general and non-specific response of the whole organism to stress. The stress might take many forms, such as trauma, surgery, exposure to severe cold, and so on. Though useful at the time, with increasing knowledge the concept has proved to be an oversimplification. Further studies were made with better techniques during the Korean war (Howard, 1955) and in recent years there has been a large study, which still continues, by F. D. Moore and his group at the Peter Bent Brigham Hospital in Boston, whose many publications have culminated in a major monograph by Moore in 1959, to which I have constantly referred in preparing this review.

To put present knowledge in a practical setting, let us consider the endocrine events in a surgical patient subjected to an operation of moderate severity, say a partial gastrectomy. The responses occur in several phases:

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Phase I: The phase of injury

This lasts two to four days. There is tachycardia, mild fever, absent peristalsis, no appetite. The muscles generally and the lymphoid tissues waste, while there is a marked loss of nitrogen in the urine, retention of sodium and water, and a urinary loss of potassium in excess of that expected from the extent of tissue destruction. A process of traumatic inflammation commences in the wound. Many hormones are concerned in these processes. It is a mistake to think that they are mainly of adrenal cortical origin.

Adrenal medullary activity begins early, with increased blood levels of the catechol amines adrenalin and noradrenalin, and increased excretion of these substances in the urine (Halme *et alii*, 1957). Fear and excitement, anoxia, ether anaesthesia, haemorrhage and oligaemic shock are potent stimuli for their secretion. A stormy ether induction therefore in an apprehensive patient will produce high blood levels, but these soon subside after average surgery, although the response from haemorrhage and shock takes longer to correct, and when there is much tissue destruction, especially in burns, the excretion may remain high for long periods (Goodall *et alii*, 1957). There is a marked sex difference in the acute response, females showing much lower levels and smaller rises than males (Halme *et alii*, 1957).

At the same time afferent pathways both nervous and humoral, but especially the former, for "wound hormones" probably play little part, stimulate the anterior median eminence of the hypothalamus in the floor of the third ventricle (Hume, 1953) which thereupon liberates a neurohumour which stimulates the anterior lobe of the pituitary to secrete ACTH. The latter is a relatively non-species-specific protein hormone with a small molecule, and in its turn it stimulates the inner zones of the adrenal cortex to secrete corticoid hormones of the hydrocortisone group. There is a "feedback" mechanism in

that the rise of blood corticoids tends to inhibit the further secretion of ACTH. This is how corticoid levels are maintained in health, but the mechanisms are somewhat upset in disease. The elegant work of Harris (1952) and of Harris and Jacobsohn (1952) has made it clear that normal adrenal size is maintained only as long as the hypophyseal portal system of blood vessels is intact. If the pituitary is transplanted to beneath the temporal lobe, even though the gland becomes well vascularized the adrenal cortex atrophies. If the pituitary is grafted back under the hypothalamus adrenal size and function are maintained. This is good evidence for the existence of a hypothalamic trophic humour controlling ACTH production. At this point we may note that adrenalin can cause some ACTH release by a direct action on the hypothalamus, but is not essential for it, because ACTH release occurs in traumatized totally sympathectomized animals (Hume, 1949). It does not occur after traumatizing a totally denervated limb (Hume, 1949).

The clinical index of increased corticoid output is the level of the circulating eosinophiles (Thorn *et alii*, 1953), and this is substantially zero by the time the incision is being sutured (Moore, 1959). ACTH can now be assayed in blood, and has been shown definitely to rise after injury. There have been many studies of blood and urinary corticoid levels after injury and surgery (e.g. Jepson *et alii*, 1956, 1957) and a general pattern is beginning to emerge. In man the normal blood 17-hydrocorticoid level (largely hydrocortisone) is 5-15 μg per cent. Within minutes of the start of surgery this rises to 30-80 μg per cent. Studies on adrenal vein blood have shown that the rise results from increased secretion rate in the gland. Again there is a clear sex difference in the response, for the female shows lower levels and a smaller rise than the male (Halme *et alii*, 1957). The excess circulating hormone is got rid of by conjugation to glucuronates, by degradation, and by conversion, probably in the liver, to 17-ketosteroids which are excreted. The peak and duration of the steroid response will therefore be affected by liver disease, by kidney disease and by circulatory failure quite apart from the more important effect of the nature and duration of the trauma. After uncomplicated surgery in a fit patient the peak of circulating steroids may be reached as early as six hours, and the level is substantially normal again in not much more than twenty-four hours (Moore, 1959). Urinary levels lag a little behind this. If complications ensue and abnormal stimuli persist, the steroids may remain elevated for a much longer period. Indeed, if the eosinophiles remain low after the sixth day a complication of some sort is likely to be present (Moore, 1959).

There is also increased secretion of the mineralocorticoid aldosterone after trauma (Llaurado, 1955; Llaurado and Woodruff, 1957) but the correlation of levels of this hormone with those of hydrocorticoids is not usually at all close. This is not surprising, because aldosterone is very largely though not entirely independent of ACTH control (Muller *et alii*, 1956) and it is probably not going too far beyond the evidence to suggest that the outer (glomerular) zone of the adrenal cortex, which is the site of aldosterone production (Greep and Deane, 1949) is essentially an independent gland which just happens to be combined with the hydrocorticoid-secreting gland and the catechol-amine-secreting gland to form a composite organ. Houssay and Sammartino showed as long ago as 1933 that the glomerular zone of the adrenal cortex does not atrophy after hypophysectomy.

Changes in sodium, potassium and body water have a profound effect on aldosterone production (*Brit. Med. J.*, 1959). While not denying the possibility that electrolyte receptors exist somewhere in the circulation, just as they exist in the taste buds, it seems clear from the elegant work of Denton and others (1959) that the stimulus for aldosterone secretion is not the sodium content of the blood perfusing the adrenal cortex, but is related to changes in extracellular fluid volume, and that sodium is effective through its influence on volume. The volume receptors are stretch receptors located probably in the wall of the right auricle and in the vena cava above and below the hepatic veins (Bartter *et alii*, 1959; Anderson *et alii*, 1959) and they send impulses via nervous pathways to a central receptor area. Very painstaking ablation experiments by Farrell (1959) combined with bio-assay of aldosterone in the cats subjected to these procedures, have furnished good evidence that this area is not located in

the hypothalamus but in the dorsal midbrain in the region of the pineal gland and subcommissural body. From this region there is elaborated a neurohumour distinct from ACTH (Bartter *et alii*, 1959; Denton *et alii*, 1959) which stimulates aldosterone secretion from the adrenal cortex. Further evidence for the existence of a soluble trophic factor derived from the head is that decapitated animals show low levels of aldosterone, but not if the carotid and jugular vessels are left intact (Rauschkolb and Farrell, 1956).

The afferent pathways to the dorsal midbrain centre are still largely unknown, but the vagus is important, particularly for stimuli which decrease aldosterone production. Vagal section does not prevent aldosterone secretion on constriction of the vena cava, but the fall in secretion after releasing the constriction does not occur when the vagi are cut (Mills *et alii*, 1958). However vagal section alone, without caval constriction, does not increase aldosterone secretion, so there does not seem to be a vagal "tone" depressing aldosterone secretion (Bartter *et alii*, 1959).

Intense research is going on in this field. An obvious thing to try is the effect of simple pinealectomy, because this organ is rich in aldosterone-stimulating activity (Farrell, 1959). Is it fanciful to wonder whether the pineal with its underlying nerve centres resembles the hypophysis and its hypothalamic centres? Again it does not seem likely that the pineal region is active merely because it is a relay station of fibres coming from above, for aldosterone secretion is not affected in animals with the brain stem transected just above the pineal. However some aldosterone-stimulating effect is constantly present in extracts made from the hypothalamus, and Farrell (1959) agrees with Liddle *et alii* (1956) and with Bartter *et alii* (1959) that there is a fraction of aldosterone secretion which is controlled by ACTH. For this reason aldosterone is *not* necessary for survival because the overlap of biological activity permits enough mineralocorticoid secretion. The location of electrolyte-controlling activity in the pineal region is interesting, and Farrell (1959) speculates that it may well be a much older system phylogenetically than the ACTH-hydrocorticoid system, and is a relic of the pineal eye of lower vertebrates, which is possibly an electrolyte receptor.

The function of aldosterone is to retain sodium in the renal tubule. In this function it is more than 30 times as active as DOCA (Desaulles *et alii*, 1953) and in the stimulated state the recall of sodium from the tubule is almost complete.

While aldosterone is influencing the renal tubule to retain sodium, the antidiuretic hormone (ADH) of the posterior pituitary is actively conserving water. Verney (1958) has summarized his many years of work on this substance. A tiny increase in plasma osmotic pressure stimulates osmoreceptors in the supra-optic and paraventricular regions of the hypothalamus to cause the release of ADH. The functioning of the hypothalamic centres is apparently dependent on intact nervous pathways between them and the paraventricular nucleus of the thalamus above them. The results of ablation experiments on the latter centre have not yet appeared, but we may regard this finding as yet another example of the movement of neurological thought away from the concept of small specific "centres" with a particular function. More and more the brain is being found to work by the co-ordinated activity of large regions, embracing many nuclei and tracts.

Verney calculates that in the dog each of the 10^5 osmoreceptor neurones releases 12,000 molecules of ADH, a relatively simple peptide of 8 amino acids, every second, or 60 per renal tubular cell. This seems a very small number to have a physiological effect. Perhaps therefore the hormone acts only on some of the tubule cells, but even assuming that all the molecules are taken up by the distal convolution alone, the ratio becomes only 500 to 1. However on the analogy of botulinus toxin, of which the toxic dose may be as small as 5 molecules per nerve cell (Robb Smith, 1957) it is not impossible that a very few molecules of ADH may have a physiological effect.

Other endocrines are less concerned in the response to surgery. Goldenberg *et alii* (1956) find increased uptake of I^{131} by the thyroid and an increase in PBI^{131} after surgery, except in those patients in whom chronic illness or recent acute trauma before the operation which he studied had already (he thought) produced an activation of the thyroid. Similarly Brown-Grant (1956) found that surgical trauma inhibited the release of

iodine from the thyroid. The blood sugar is also elevated after surgery, both by sympatho-adrenal stimulation and by the increased secretion of glucocorticoids. There is also, we may note, a very rapid burning of liver glycogen as well as of muscle glycogen in the post-operative patient, so that the blood sugar changes rapidly. The parathyroids are not much affected by average surgery. The function of the gonads is depressed in the early phases, and interest in sexual matters does not return until well into the convalescent period.

Turning now to the metabolic consequences of the increased endocrine activity after trauma, perhaps the most remarkable is the very great loss of nitrogen in the urine. This is derived mostly from muscle, and is excreted as urea, a freely diffusible and electrically negative crystalloid. The amount of nitrogen lost is less in starved persons, and may be somewhat reduced but not prevented by giving large amounts of nitrogen post-operatively. The latter procedure is analogous to keeping up the water level in a leaky bucket; the water runs out almost as fast as it is poured in. The cause of the negative nitrogen balance is not at all clear. Certainly starvation and immobilization play their part, for they will produce a negative nitrogen balance by themselves, and ACTH and Cortisone have also been shown to produce a loss of nitrogen in the urine (Li *et alii*, 1949). Cuthbertson (1945) calculated that after a leg fracture as much as 137 g. of nitrogen, derived from 856 g. of protein, could be lost in this way. This is an amount of protein 3 to 4 times the total protein content of the liver. Cuthbertson suggests that the "purpose" of this great protein mobilization is to provide a mechanism for healing independent of food supply, and to flood the bloodstream with the essential building bricks for tissue repair. One of the most important of these is the sulphur-containing amino acid methionine, for this is the only amino acid which will by itself reduce the nitrogen loss after trauma.

Liver and muscle glycogen are quickly burned after injury. As protein is degraded some of the fragments are burned. Even so, the chief source of energy after trauma is fat. If 200-500 g. of fat are oxidized daily they provide 2,000-4,500 calories and 200-500 cc. of water of oxidation, which must therefore be added to the "intake" side of the fluid balance chart.

Phase II: The turning point

In straightforward surgery this occurs at about the third to seventh day and lasts for one to two days. In more complex cases it may be long delayed or obscured. The patient feels better. The wound is less painful and fever is less or vanishes. Appetite and bowel functions return. After the initial phase of demolition in the wound, which is perhaps aided by the ability of the steroids to prevent premature collagen deposition, but retarded by their property of preventing leucocyte migration (Ketchel *et alii*, 1958) the wound begins to acquire tensile strength. The substances most essential for this process are methionine and vitamin C.

The excessive adreno-medullary secretions have long since returned to normal. If they have not, the turning point will not be reached until they do. Adrenal cortical secretions also now return to normal rates and levels. The urinary 17-hydroxycorticoid excretion falls, and the circulating eosinophile count often rises at the same time. Urinary nitrogen and potassium loss decreases, and the balances become positive. Sodium excretion rises, and there is often a water and salt diuresis, although it usually correlates poorly with urinary steroid excretion. Upswing of potassium on the chart regularly precedes that of nitrogen. Thus it is most important to provide an increasing and adequate diet during this phase. Protein synthesis cannot occur unless the intake is adequate to provide a positive nitrogen balance. If this is achieved, the lean tissues can gain about 90 g./day (Moore, 1959).

Phase III: Repair of muscle strength

The continuing addition of nitrogen as muscle resembles normal growth, and in children skeletal growth returns. The endocrine setting for surgical anabolism is unknown. Presumably growth hormone (somatotrophin) is somehow involved, but we know little of its action, and patients recover from total hypophysectomy without the addition of any growth hormone, although they require corticoids to survive. Insulin, corticoids, thyroxin and gonadal hormones probably also play their part, although Moore denies that testosterone, which is anabolic, helps the normal anabolic patient.

Phase IV: Fat gain

Weight gain occurs over a long period without gain in water, which means gain in fat. However the wound remains somehow a special area for a long time. It will break down in severe scurvy, and infections tend to localize in it.

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OBSERVATIONS ON A POLYCYSTIC KIDNEY BY MICRODISSECTION*

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THE greater part of the literature on cystic kidneys deals with presumed aetiology, to a less extent morphogenesis and least with basic morphology. Speculations on causes and modes of formation have strongly influenced attitudes about these conditions at all levels of investigation; what is required, however, is a sufficient number of observations on the basic structural changes.

The relatively few examples of cystic kidneys that have been studied closely show that there are many varieties. These comprise various permutations and combinations of a comparatively few forms of parenchymal change. Since these can be elucidated only by collections of morphological studies, which can ultimately be the basis of aetiological and pathogenetic assessments, this study is presented.

The results of microdissection of a polycystic kidney from a baby aged forty-seven days are described in detail. The tubular elements were greatly shortened, showed overall swelling as well as localized cystic dilatations in the various nephron components. The description and measurements of nephrons from a normal individual of the same age period are given for comparison.

MATERIAL

The specimen was obtained post-mortem from a female baby aged forty-seven days. It had been described at birth as "small", and "ill-nourished" with "renal" facies. It was unable to suck, requiring gavage feeding. Its condition gradually deteriorated.

External examination showed a long narrow head with narrow forehead, ears set low near the angle of the jaw, broadened nose and prominent epicanthic folds. The abdomen was soft and distended.

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Apart from the renal tract the internal organs were normal. The right kidney was small, measuring 2.6 x 1.8 x 0.8 cm. with numerous tiny cysts on the cut surface. The left kidney, measuring 3 x 2 x 1 cm., contained several large cysts which almost completely replaced the parenchyma. There was a minimal amount of renal tissue in part of which there were small cysts. On the left side there was a partial stenosis of the proximal part of the ureter and some dilatation of the pelvis. A well-developed urachus was attached to a small bladder (Fig. 1).

FIG. 1. Photograph of the specimen displaying the condition of both kidneys; there are small cysts in right and large cysts in left kidney.

A specimen of a kidney from a baby of forty-nine days, obtained post-mortem, showing no gross nor histological abnormality, was also studied.

FIG. II. Low power photomicrograph of a section of right kidney cortex. Acidophilic amorphous substance is present in the lumina of many tubules. Haematoxylin and eosin. (x 45)

FIG. III. Low power photomicrograph of a section of right kidney showing area of inflammatory infiltration in lower cortical area. Infiltration is by small round cells, numerous fibroblasts and occasional polymorpho-nuclear leucocytes. Haematoxylin and eosin. (x 45)

METHODS

Appropriate pieces of tissue from both kidneys were removed for histological examination; the bulk of the tissue was used for microdissection. The material was found, by experiment, to require incubation for fifty-five hours at 22°C. in concentrated hydrochloric acid following the technique of Oliver, MacDowell and Tracy (1951). After incubation, the material was dissected by hand under a stereoscopic microscope, working at magnifications of 50 and 100. The material, removed from the dissection dish, was mounted in water on a glass slide. Photographs of microdissections are of unstained material, made with a Leica camera and adapter using 35 mm. Ilford Micro.Neg.Pan film.

FIG. IV. Low power photomicrograph of a section from left kidney. The tissue is situated between two cyst walls. Haematoxylin and eosin. (x 45)

The material from the normal kidney was treated similarly but was incubated for twenty-eight hours at 19°C.

RESULTS

Histological examination

The right kidney had greatly reduced parenchyma which showed considerable structural variety, most components being distended to some degree (Fig. II).

The renal corpuscles showed well-developed tufts of different sizes ranging from those filling the capsule to small knobs; there was thus considerable variation in capsular space. Some were cystic but these were not conspicuous.

Tubules varied considerably, from those lined by large acidophil cells to others with cuboidal darkly staining cells. Although some of the former were proximal convoluted tubules, most others were difficult to allocate in the nephron. Collections of small tubules occurred in localized groups; their lining cells usually stained deeply (Fig. III).

Dilatation of tubules occurred throughout the cortex, extending to and being prominent near the cortico-medullary junction. In some parts the dilatation involved a length of tubule but sacculations with abrupt transition from a dilated to narrow lumen occurred.

Cysts were visible through the cortex, particularly in the deeper layers. They were lined by a single, regular layer of cuboidal cells; larger cysts were lined by cuboidal to flattened cells. Some areas showed collections of small round cells, fibroblasts and occasional polymorphonuclear leucocytes (Fig. III). Thickening of vessels was pronounced in some parts.

In the more grossly cystic left kidney, vascular fibrous tissue surrounded and lay between the large cysts (Fig. IV). In this were collections of tubules containing an acidophilic homogeneous material and lined

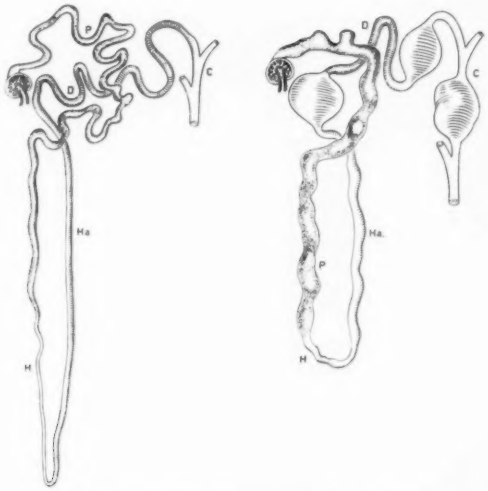

FIG. V. Diagrammatic representation of a normal (8th post-natal week) and a cystic nephron (7th post-natal week). The abnormal nephron shows the average structural features. It was significantly shorter, with slight enlargement only of the renal corpuscle, showed dilatation of all tubular components, loss of convolution and diverticula in the proximal convoluted tubule, did not have the normal narrowing of the loop of Henle, and showed local cystic dilatation and cyst formation at the apex of the ascending limb of Henle, in the distal convoluted tubule, and in the collecting tubule.

Other features described and illustrated in photomicrographs occurred less commonly.

P = proximal convoluted tubule; H = descending limb of loop of Henle; Ha = ascending limb of loop of Henle; D = distal convoluted tubule; C = collecting tubule. The two drawings are to scale from measurements made (Table 1).

by small darkly staining cells; some showed irregularly distributed stratification of the cells. Occasional glomeruli, of a "primitive" type, occurred amongst the tubules. Most of the tubules were distended and small cysts, lined by cuboidal cells, occupied most of the tissues. In some regions there were accumulations of small round cells, plasma cells and fibroblasts.

Special stains used were: Alcian blue — negative result, PAS — positive with respect to the material within the tubule lumens and Picro Gomori which showed a moderate amount of collagen throughout the tissue.

FIG. VI. Microdissection photomicrograph of an extremely short distended proximal convoluted tubule.* Renal corpuscle: diameters 140μ x 140μ; tubule: maximum diameter 140μ, minimum diameter 35μ. Length of proximal tubule: 1.6 mm. (x 100)

Microdissection examination

The maceration time required for this material was longer than expected considering the amount of connective tissue evident on histological examination; at 22°C. it was approximately twenty hours longer than anticipated and even after fifty-five hours maceration the connective tissue was still strongly adherent to the parenchyma. During dissection, special care was necessary in teasing out the nephron components as many portions were rigid, flattened, translucent and easily damaged during manipulation.

*Diverticula are present (arrows).

The renal corpuscles showed considerable variation in size and in shape and form of tuft. The diameters ranged from 75μ to 200μ, but most lay within a range of 110μ to 140μ. The diverse shapes included spherical, ovoid (Figs. VI, VII and VIII) and occasional reniform types. The tuft, in most instances, filled the capsule but, in some, it was smaller so that glomeruli exhibited a wide subcapsular space.

FIG. VII. Proximal convoluted tubule from renal corpuscle to loop of Henle. Renal corpuscle: diameters 200μ x 120μ; tubule; maximum diameter 105μ, minimum diamter 35μ. Length of proximal tubule: 2.8 mm. (x 120)

The proximal convoluted tubules were rigid and difficult to manipulate. They could not be unravelled; although less convoluted than normal, kinks appeared to be a permanent feature and tension exerted to "position" them caused fracture of the tubule. For this reason, in many instances, only small portions could be photographed. They showed great variety. The tubule was attached to the glomerulus by a neck; this was a thick structure (Fig. X), a fine thread, of such dimensions that no actual connection was obvious

(Fig. IX) or less frequently a "swan-like" type (Fig. VII). Tubular diameters ranged from approximately 30μ to 180μ and in a single length varied from 35μ to 140μ (Fig. VI). However, the average variation was between 35μ and 105μ.

There was, in addition, an over-all lack of convolution in this part of the nephron. At times, the convolution was almost absent (Figs. X and XI); when compared with a normal proximal convolution (Fig. XII) the contrast was obvious.

Diverticula, randomly distributed, occurred both singly and in a multiple arrangement (Fig. VI). They were present in the area adjacent to the glomerulus, in the first two-thirds of the tubule or, rarely, in the terminal segment.

The length of this tubule was measured and recorded (Table 1). The frequency of these readings in the lower range is probably due to the better chance of dissecting out entire segments in shorter structures.

TABLE 1

COMPARISON OF DIMENSIONS OF CYSTIC KIDNEY WITH NORMAL KIDNEY

Nephron Component	*Av. diam. in μ*	*Range*		*No. of Readings*	*Length in mm.*	*No. of Readings*
		Extreme	*Common*			
Renal Corpuscle (a)	110	60-160	100-130	80	—	—
(b)	130	75-200	110-140	30	—	—
Proximal Convoluted Tubule (a)	35	20- 60	30- 40	510*1	3.5	40
(b)	80	30-180	35-105	66*2	2.2	10
Loop of Henle (a)	12	10- 15	—	80	1.0	5
(b)	20	15- 30	—	5	So ill-defined not measured	
Ascending Limb of Henle (a)	20	15- 30	20- 25	18	2.0*3	6
(b)	40	10- 85	30- 35	20	1.5*4	6
(c)	255	95-900	—	32	—	—
Distal Convoluted Tubule (a)	25	20- 35	—	6	1.1	3
(b)	45	25- 95	—	6	1.3	3
(c)	170	112-245	—	6	—	—
Collecting Tubule near to distal convoluted tubule (a)	35	25- 50	25- 40	13	—	—
(b)	40	30- 45	—	6	—	—
(c)	220	85-425	—	5	—	—
Complete Nephron (a)	—	—	—	—	7.1 5.9 5.8	—
(b)	—	—	—	—	3.1	—

Key: (a) Normal baby, aged 7 weeks.
(b) Polycystic kidney — dimensions excluding local cystic dilatations.
(c) Polycystic kidney — dimensions of cysts.
*1 40 proximal convoluted tubules measured.
*2 25 proximal convoluted tubules measured.
*3 Range = 1.2-2.7 mm.
*4 Range = 0.7-2.0 mm.

The normally thin descending loop of Henle was short and stout in this kidney, and often difficult to distinguish from proximal convoluted tubule. Diameters recorded ranged from 15μ to 30μ. The ascending limb of Henle's loop also departed grossly from normal. The length of this limb was, as the other parts of the nephron, much reduced in length, with great variation in diameter. This, apart from an overall dilatation in the limb itself, was exaggerated in a cystic type of dilatation occurring, without exception, in the region of, or at a level with, the macula

FIG. VIII. Proximal convoluted tubule showing poor convolution and variable diameters of tubule which is joined to the glomerulus by a slender neck. Glomerulus shows a thickened arteriole with Ludwig's vessel. Renal corpuscle: diameters 150μ x 95μ. Length of proximal tubule: 1.7 mm. (x 145)

densa. Here, many varieties of cyst, and combination of cysts, were present (Figs. XIII-XVII). In rare instances cysts were observed to end blindly (Fig. XIV). Occasionally, in addition, cystic structures also occurred in the limb near the crest of the loop. Diameters of the tubule itself ranged from 10μ to 85μ, whilst cystic dilatations were from 95μ to 900μ; diverticula also were observed frequently here. The nephron components in this region appeared to be translucent and flattened.

The distal convoluted tubule was also dilated (Fig. XVII), though not reduced in length, and showed localized, cystic dilatations. The collecting tubules were usually prominent as they contained much dark material within their lumen (Fig. XVII) and also included local cystic dilatation (Fig. XVIII).

FIG. IX. Complete proximal convoluted tubule showing poor convolution and dilatation in the tubule. Renal corpuscle: diameters 120μ x 120μ. Tubule diameter: proximal to glomerulus, 105μ; at distal end 30μ. Length of proximal convoluted tubule: 2.6 mm. (x 110)

No significant change was observed in the larger blood vessels, but in the smaller ones there was some thickening of the vessel wall. The afferent arterioles were generally thickened (Figs. VIII and XI), while Ludwig's vessel was often observed (Fig. VIII); in addition, the efferent arteriole was dissected out easily.

Tissue from the left kidney showed occasional portions of proximal convoluted tubule but it was not possible to identify the remaining remnants with certainty. The proximal convoluted tubules were poorly convoluted, varied considerably in tubular diameter and

showed diverticula. A black substance, released by the acid maceration and not removable by washing, enveloped the tissue and remained firmly adherent during dissection.

The dimensions of normal nephrons are given in Table 1. On the basis of these findings, a typical normal nephron and a composite from the polycystic kidney have been reconstructed in the drawings (Fig. V).

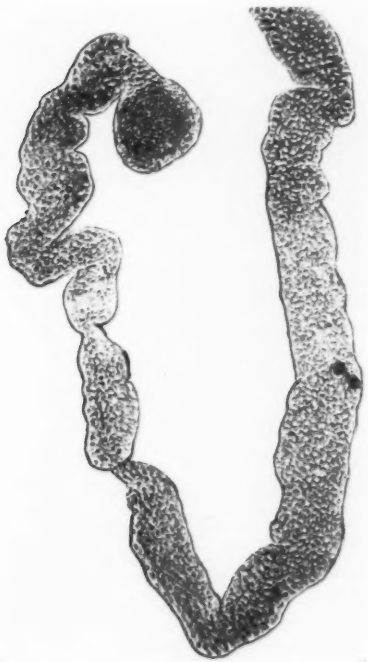

FIG. X. Flattened, dilated, pale length of proximal tubule showing poor convolution. Diameters of renal corpuscle: 140μ x 120μ; tubule: maximum diameter 105μ, minimum 40μ. (x 100)

DISCUSSION

During the last hundred years, much has been written on renal cysts but a review of this shows that most statements deal with speculations regarding aetiology. These clearly do not have an adequate foundation of observation. Some experimental work has been done (von Mutach, 1895; Pettersen, 1903; Tollens, 1904; Bagg, 1925) but with almost consistently negative results. It is apparent that our present knowledge is still too incomplete to make this an immediately fruitful field.

With the growth of knowledge and appreciation of pathological principles, conditions were recognized as being due to extrinsic or intrinsic factors. Virchow (1869) postulated foetal papillitis in which the results of connective tissue proliferation and subsequent contraction caused atresia of the renal papillae, tubular occlusion and proximal dilatation, thus giving rise to polycystic disease. On the other hand von Mutach (1895) suggested that a partial failure of growth of components of the ureteric bud, with non-union of some of these with glomeruli of the renal anlage, resulted in the cyst formation. The rapid development of knowledge of embryology at the period made this the generally accepted view. Kampmeier (1923) considered that "primary uriniferous tubules" failed to develop normally and a similar point of view was supported by McKenna and Kampmeier (1934) and Norris and Tyson (1947). In 1933 Beeson regarded cystic change as an expression of a deficiency of the renal anlage and even more recently Roos (1941) stated this as "a developmental disturbance which may be attributed to defective germ plasm". Gordon and Trasoff (1937) considered the parenchymal changes to be secondary to vascular disturbance — "obliterative changes in the arterioles and small arteries of the kidney". Support for the general idea that peculiarities of development are, in some way, responsible was found in the association with abnormalities in other organs (von Mutach, 1895; Bunting, 1906; Maeder, 1907; Roos, 1941). Furthermore, a familial incidence has been observed (Pettersen, 1903; Bunting, 1906; Fuller, 1928-29; Gordon and Trasoff, 1937). However, these suggestions are not sufficiently precise to help understanding of the condition.

Morphogenesis is, at present, much more susceptible of analysis and critical examination. Virchow (1869) considered tubular obstruction to be the process involved; however, it is now recognized that this alone is more likely to produce atrophy than cyst formation. Kampmeier's (1923) view that an original cystic change in some developmentally predisposed nephrons (due to speculative factors) would then, by way of cystic growth

and pressure on adjacent units, result in a type of chain reaction with production of more cysts, is untenable with present-day understanding of cell relations. The suggestion of von Mutach (1895) and Roos (1941) that non-union of developmental components could produce cystic changes in the proximal segment also does not deal adequately with the fundamental processes involved. Indeed, the "failure of growth" does not explain what is, in fact, an increase in tissue. On the other hand, Beeson (1933) emphasized the important features of epithelial hyperplasia in the cyst wall and Bricker and Patton (1955) observed that the Na^+, Cl^- and K^+ and total soluble concentrations in polycystic fluid frequently exceed plasma levels. This indicates functional activity of the cells lining the cysts and thus an active as opposed to a passive process. Gruenwald (1947) remarked that "many different causes and even entirely different developmental mechanisms may produce very similar end results" and he emphasized and elaborated the concept of "organizer action". This, however, is still too vague and speculative to be of direct assistance in elucidating the mode of formation of the cysts.

Fig. XI Fig. XII

FIG. XI. Complete proximal tubule showing poor convolution. Length of tubule: 2.2 mm. (x 60)

FIG. XII. Proximal convoluted tubule (dissected from normal kidney — baby aged 7 weeks). Renal corpuscle: diameters 110μ x 100μ. Tubule: diameters ranged from 25μ to 50μ. Length of proximal tubule: 3.6 mm. (x 70)

The investigations on which the large superstructure of hypothesis is built range from direct study of tissues — the simple observation of the embryonic rather than the inflammatory type of tissue present (von Mutach, 1895; Kampmeier, 1923) — to the reconstruction of nephrons from serial sections (Roos, 1941; Beeson, 1933); and chemical comparisons of cyst fluids and serum levels of sodium, chloride and potassium (Bricker and Patton, 1955) provide valuable information here.

Although it was at first thought that cysts arose in glomeruli (Roos, 1941), Lambert (1947) from reconstructions of serial sections, demonstrated cysts as glomerular, tubular and excretory types. This has been confirmed by microdissection (Bialestock, 1956, 1958, 1960). It has been demonstrated that there is a considerable range of change

FIG. XIII. Upper portion of ascending limb of loop of Henle, and distal convoluted tubule, both showing gross dilatation. Diameter of tubule: maximum 245μ, minimum 30μ. (x 100)

from the extremes of giant nephrons (Bialestock, 1956) to the short units seen in the present study. The variety of types of cysts indicates that it is fruitless to attempt to deduce histogenesis of polycystic disease as a whole from the findings in a single kidney. At the same time it appears that there are several well defined types of cystic change, differing in degree, distribution and extent. To what extent these various types occur consistently still remains to be determined.

FIG. XIV. Short, flattened length of ascending limb of Henle's loop, ending blindly in a cystic dilatation. A diverticulum is present on the narrow length of the tubule. Maximum diameter: 245μ. (x 100)

FIG. XV. A cyst, attached to tubule (ascending limb of Henle's loop) by two small connections, one of which was broken in mounting. Cyst: 660μ x 520μ. (x 95)

FIG. XVI. Cystic dilatations at apex of ascending limb. There appear to be two or three confluent dilated areas. Diameter of tubule proper: 42μ; at dilated areas: 255μ. (x 95)

FIG. XVII. Illustration of the relationship of the different nephron components. This was one of the few examples seen where the cystic dilatation at the apex of the ascending limb of Henle's loop was not prominent. In manipulation, the proximal convoluted tubule was almost severed from its renal corpuscle. Dark material is visible within the lumen of the distal convoluted tubule and collecting tubule. (x 100)

Of the host of speculations advanced, and in the light of present-day biochemical knowledge, a chemical factor through the medium of organizer, enzyme or hormone appears to be the most plausible argument yet presented. At the same time, what is required is a sufficiently large number of observations from different cases to indicate the susceptible parts of the nephron and the differential susceptibility at different stages of development with, presumably, different types and intensities of stimulus. The present study provides one small addition to this pool of information.

FIG. XVIII. Thin-walled cystic dilatation in a collecting tubule. Cyst was entire but torn in dissecting away from massive connective tissue. Maximum diameter in dilated area: 425μ. (x 100)

SUMMARY

An example of cystic kidney in a baby of forty-seven days has been examined by microdissection. There was some general dilatation of a shortened nephron and this was most prominent (with cyst formation) in the region of the junction of ascending limb of

the loop of Henle with the distal convolution, other cysts occurring in the distal convoluted and collecting tubules.

Although a few examples of cystic renal disease have been described in some detail, these still remain as independent and almost unrelated observations. The failure to provide an adequate classification of morphological types is due to the paucity of information on nephron structure in cystic kidneys. When sufficient have been described some classification, which can then be a basis for other investigations, will be practicable.

ACKNOWLEDGEMENT

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MALIGNANT SPINAL EXTRADURAL TUMOURS*†

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COMPRESSION of the spinal cord by a malignant extradural neoplasm may be the initial feature of a hitherto unsuspected or unrecognized malignancy. In these cases surgical intervention will nearly always be required in order to clarify the diagnosis. On the other hand it may present as a terminal symptom in an advanced and moribund case of malignant disease. Surgical treatment is clearly not indicated in such cases and doubtless many such patients are not referred to a neurosurgical clinic. Between these two extremes there is a considerable group of patients with a reasonably well-controlled malignant disease, who develop paraplegia from metastatic spinal deposits. It is in this group that most of the problems of surgical management arise: this paper attempts to answer some of these problems by a study of the pathology, diagnostic features and prognosis of this type of spinal tumour. The material consists of 18 cases of metastatic spinal tumour from the files of the Neurosurgical Clinic, Royal Adelaide Hospital.

Comparatively few studies have been made of the short-term prognosis of malignant spinal tumours and the indications for and results of decompression of the spinal cord in such cases have been similarly neglected. Törmä (1957) made a retrospective study of 250 cases of malignant spinal tumour from Scandinavian neurosurgical clinics and provided a detailed account of the clinical and pathological features of such tumours. However, his report lacks any details of neurosurgical follow-up after surgical treatment and his conclusions, based on survival only, that surgery is valueless in these cases, cannot go unchallenged when a wider view is taken of the results of surgery particularly in regard to recovery from paraplegia.

Other writers, for example, Perese (1958) and Rogers (1958) advocate laminectomy and decompression in all cases of progressive spinal cord compression. The cases to be considered below show that this policy of operative treatment can achieve satisfactory results, irrespective of the nature of the tumour, provided only that diagnosis is made and treatment undertaken at the earliest possible moment.

Pathology

These tumours are always found to lie in the epidural space. They may arise there initially or invade the epidural space from primary involvement of the vertebral body or neural arch. There is a decided tendency to longitudinal spread in the epidural space, a feature demonstrated in exaggerated form by Case 3 in which the tumour extended from the upper thoracic region to upper lumbar region. Usually, however, the extent of the tumour at the time of producing signs of cord compression overlies two or three vertebrae. By contrast there is surprisingly little tendency for the tumour to spread through the intervertebral foramina into the extraspinal tissues in the way that some benign tumours, such as neurofibroma and haemangioma may do with the production of dumbbell tumours. This is doubtless merely a manifestation of the more rapid mode of growth of malignant tumours so that they spread along the tissue plane of least resistance, viz., the epidural space.

It is also to be noted that penetration of the dura by a metastatic neoplasm is rare: there were no examples in this series, although Case 1 illustrates the reverse and equally uncommon process of transdural spread of a malignant intradural ependymoma.

The explanation of the curious predilection of these metastases for the epidural space is

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to be found in a consideration of the vertebral system of veins. This extensive longitudinal network of valveless veins was brought into prominence by Batson (1940, 1957) and Herlihy (1947) (Fig. I).

provided slight abdominal compression was applied during injection so as to favour a blood flow from the caval venous system to the vertebral veins. Under these circumstances metastasis to the lungs was un-

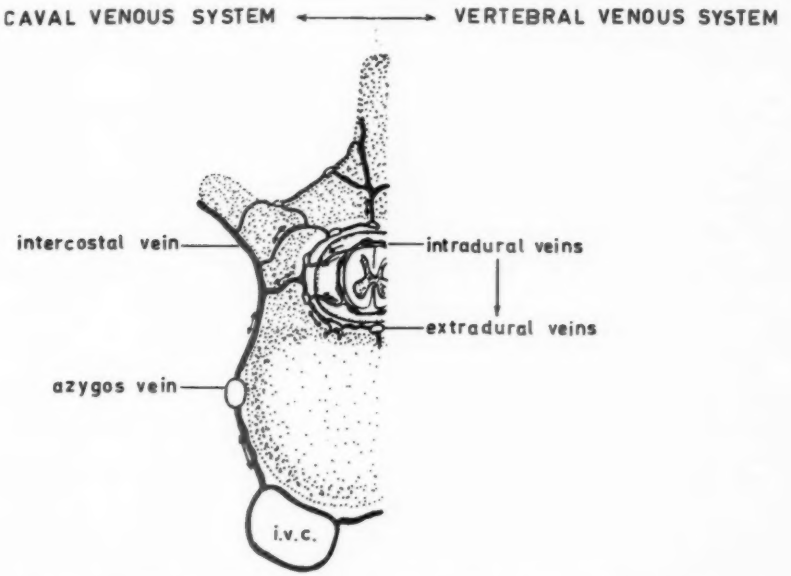

FIG. I. Illustrating communications between the vertebral venous sytem and the caval venous system. Note the unidirectional flow in the intradural veins and the bidirectional flow elsewhere.

The vertebral venous plexus communicates superiorly with the veins of the head and neck, including the intracranial venous sinuses via the emissary veins, and inferiorly with prostatic and pelvic floor veins, whilst throughout the spinal column there are regular valveless connections with the inferior and superior venae cavae via the intercostal and azygos veins. This extensive system of veins makes possible the spread of tumour emboli into the epidural space from primary tumours situated anywhere within the body, although naturally those tumours lying along the lines of communication into the vertebral venous system, e.g., prostatic carcinoma, are advantageously situated to metastasize to the spinal canal. Coman and de Long (1951) have demonstrated convincingly in rats and rabbits that tumour cell suspensions injected into the femoral veins passed directly into the vertebral veins producing, almost always, vertebral metastases common, but when the injection was made without abdominal compression metastases almost invariably appeared in the lungs only. Some such mechanism as this, e.g., coughing or straining at stool, may determine the entry of tumour emboli directly into the vertebral veins in man.

The rarity of metastases in the spinal cord itself requires explanation. This is partly due to the powerful biological resistance of the dura to direct local invasion. Anyone who has had any experience with extradural spinal tumours must have noticed how it is frequently possible to peel the tumour off the dura with practically no dissection as if the dura were a completely inert membrane, a property which has been emphasized by Rogers (1958). On the other hand there are emergent veins at almost every segmental level connecting the spinal cord veins with the vertebral venous plexus and without some

special haemodynamic arrangement one would expect blood-borne metastases to the spinal cord to be almost as common as extradural metastasis. Batson, quoted by Coman and de Long (1951) considers that acute angulation of the spinal segmental veins where they leave the main spinal cord veins is a mechanical hindrance to the deposition of tumour emboli within the spinal cord. An equally likely mechanism, I suggest, is that the pressure within the spinal cord veins is considerably higher than that in the extradural vertebral venous plexus. The pressure within the spinal cord veins must of necessity be slightly higher than the cerebrospinal fluid pressure, otherwise the veins would collapse. In the lumbar and thoracic region the C.S.F. pressure is upwards of 100 mm. of water in the recumbent position. Any rise of pressure in the caval venous system is immediately transmitted to the C.S.F., the pressure of which rises accordingly, a fact which may be readily demonstrated by anyone who takes the trouble to test the effect of abdominal compression or coughing whilst carrying out spinal manometry. It is likely therefore that the intradural venous pressure is always higher than the pressure in the vertebral and caval venous systems and that the venous flow in the spinal theca is either axial or centrifugal but seldom centripetal, thus preventing intradural transmission of tumour emboli by this route (Fig. I).

In Case 3 (extradural liposarcoma) at autopsy there was almost complete embolization of the vertebral venous plexus. The extradural space, including the vertebral veins, was occupied by tumour from at least the 3rd thoracic to 2nd lumbar vertebra and many of the communications between the vertebral and the caval venous systems, such as the intercostal and lumbar veins and the venae azygos were converted into solid cords of tumour. There was also a tumour embolus in the right cavernous sinus. In spite of this extensive involvement of the extradural veins there was no sign at autopsy of intradural

TABLE 1

ANALYSIS OF 18 CASES OF MALIGNANT EXTRADURAL SPINAL TUMOUR

Serial No.	*Sex*	*Age*	*Duration of prodromal symptoms*	*Duration of cord symptoms*	*Neurological status at time of operation*	*Histopathology*	*Result*
79	Male	22	3 months	3 months	Partial cauda equina lesion	Ependymoma	Good—survived 5 years from 1st operation
143	Male	38	18 months	3 weeks	Partial paraplegia—T.5	Hodgkin's disease	Good—survival 16 months
244	Male	21	6 months	3 days	Complete paraplegia —L.3	Liposarcoma	Poor—Laminectomy L.2—T.3 —no recovery. Survived 1 month
210	Male	69	8 months	1 month	Partial paraplegia—T.5	Prostatic carcinoma	Poor—No recovery. Survived 3 weeks
295	Male	77	5 months	2 months	Partial paraplegia—T.5	Prostatic carcinoma	Good—Excellent recovery—normal ambulation
289	Female	42	9 months	12 days	Partial paraplegia—T.3	Bronchial carcinoma	Good—Unaided ambulation
414	Female	29	6 weeks	3 days	Partial paraplegia—C.8	Lymphatic leukaemia	Poor—Survival 5 months
765	Female	31	2 months	5 days	Partial paraplegia—T.6	Malignant melanoma	Fair—Good initial result. Recurrence of paraplegia 6 weeks later. No recovery following further laminectomy
665	Female	61	7 months	1 month	Partial paraplegia—T.2	Breast carcinoma	Good—ambulant
558	Male	75	6 weeks	1 month	Partial paraplegia—T.2	Myeloma	Poor—Died 5 days post-operatively from intestinal obstruction
701	Female	46	3 months	8 days	Partial paraplegia—T.4	Breast carcinoma	Good—Ambulant-survival 4 months
062	Male	54	3 months	3 weeks	Partial paraplegia, T.4 and L.2	Prostatic carcinoma	Fair—Improved motor power. Died pulmonary embolus 14 days post-operative
890	Male	63	3 months	5 day	Complete paraplegia —T.3	Reticulosis	Poor—Survival 6 weeks
1069	Male	43	4 months	3 days	Complete paraplegia —T.2, 6 hours	Reticulosis	Fair—Good initial improvement then relapsed
0589	Female	70	1 month	9 days	Incomplete T.2 paraplegia	Unidentified carcinoma	Fair initial improvement
1116	Female	44	3 months	4 days	Complete L.3 cauda equina lesion—24 hr.	Brill-Simmonds disease	Fair—Improved motor function
0550	Male	43	4 weeks	2 weeks	Incomplete cauda equina lesion	Lymphosarcoma	Fair—Initial good recovery with relapse 2 months post-operative
1035	Male	69	5 months	24 hours	Complete paraplegia —T.12	Prostatic carcinoma	Poor—no recovery

spread of the tumour, a testimony to the efficacy of the biological barrier of the dura and to the centrifugal flow of blood in the intradural veins.

CLINICAL FEATURES

The salient clinical features in 18 cases of malignant extradural spinal tumour are set out in Table 1. Although the end result in all untreated cases is the same, viz., paraplegia from compression of the spinal cord or cauda equina, the natural history of this type of tumour can conveniently be divided into two stages, the stage of prodromal symptoms and the stage of spinal cord symptoms.

(a) *Prodromal stage*

Pain is the outstanding and often the only symptom of this period. The pain may be of two types, local spinal pain or root pain. Local spinal pain is usually constant and aggravated by exertion and often accompanied by local tenderness and deformity (kyphus) of the involved vertebrae. Pain of this type us usually due to neoplastic involvement of the vertebral body or neural arch and characteristic changes may often be seen in plain X-rays of the spinal column. Root pain is due to strangulation of the emergent nerve roots in the intervertebral foramina. It has the usual features of segmental root pain but, in addition, there may be wasting and muscular weakness of segmental distribution. This muscular weakness is only likely to be evident if the neoplasm involves nerve roots running to the brachial or lumbosacral plexuses.

The important feature of either type of pain is that it may, and often does, persist for weeks or months without any additional signs until paraplegia develops. The absence of other symptoms and signs results in the true nature of the pain being misinterpreted in practically every instance. Even when the patient is known to be harbouring a malignancy this prodromal pain is frequently passed off as being of no consequence or due to fibrositis or spondylosis. In this series prodromal spinal or root pain had been present for at least four weeks in every instance and as long as eighteen months in one case before the onset of cord signs. Examination of the cerebrospinal fluid in this stage may reveal a considerably increased protein content and thus point to the necessity of further investigations, as for example in Case 9 in which the C.S.F. protein was 150 mgm. per 100 ml. When signs of cord compression have developed lumbar puncture must be done with caution for fear of accelerating the cord signs, and is preferably combined with myelography so that immediate surgical treatment can be carried out if necessary. The hazards of lumbar puncture carried out as a "routine" procedure in such cases is exemplified by Case 2 in which lumbar puncture was performed when the patient had only slight weakness of the legs for the preceding three weeks. Over the next forty-eight hours after lumbar puncture this progressed to an almost complete paraplegia.

Törmä (1957) also comments on the hazards of indiscriminate lumbar puncture and records four cases in which paraplegia occurred immediately following lumbar puncture; 16 cases in which paraplegia occurred within two days of lumbar punctures; and five cases in which there was a sudden increase in paraparesis after lumbar puncture.

(b) *Stage of cord compression*

Whereas the prodromal stage of local pain may be prolonged, the stage of cord compression is usually short and complete paraplegia develops rapidly. In this series the duration of cord signs before operation ranged from one day to three months and in 16 cases (90 per cent.) the period was one month or less. At the time of operation the neurological lesion was severe in every case amounting to an absolute paraplegia in 5 cases (28 per cent.) and a complete motor paralysis with sensory sparing in 4 cases (22 per cent.), i.e., in 50 per cent. there was complete motor paralysis.

The diagnosis can be confirmed in every case by radiological examination of the spine including myelography. In 12 cases (66 per cent.) plain radiographs showed diagnostic changes of erosion of pedicles or collapse of vertebral bodies. In this event operation can be undertaken forthwith, although one may occasionally be misled both radiologically and at operation by the low-grade osteitis of the vertebral column resulting from chronic urinary tract infections.

Case report

R.A.H. 12133/59. R.B., male, aged 58 years, complained of backache for three months following an alleged attack of pneumonia. Five days before admission he had developed a severe paraplegia with sensory loss to T.5. X-rays of thoracic spine showed wedging of T.3 and T.4 with narrowing of the disc space. Myelography showed complete arrest at this level from anterior extradural compression. Exploratory laminectomy was undertaken on 11th February, 1959, and a mass of tissue was found compressing the cord anteriorly. No pus was encountered and the lesion was thought at first to be a reticulosis. The patient died suddenly and inexplicably two days after operation and autopsy showed an unsuspected chronic pyelonephritis with low-grade osteomyelitis of the spine, and extradural pyogenic granuloma.

If plain films of the spine appear normal, myelography will confirm the presence, location and type of obstruction of the spinal theca. If lumbar puncture has not been performed previously, myelography should be done at the time of this initial lumbar puncture in order to establish the diagnosis as soon as possible and to avoid the deterioration which may occur following lumbar puncture. Typically, there is a transverse or tapering arrest of the contrast medium without any filling defect and, in every case in this series in which myelography was performed, the obstruction to the contrast medium was complete.

Myelography will also exclude a compression lesion of the cord and thus substantiate a clinical diagnosis of carcinomatous myelopathy of the cord which otherwise may simulate a cord tumour exactly.

Case report

R.A.H. 8748/59. I.E., male, aged 52 years, referred by Mr. S. Krantz. Two weeks before admission to the Royal Adelaide Hospital he began to drag his right leg and simultaneously developed right sciatic pain. Three days before admission he developed retention of urine necessitating repeated catheterization, and at the same time he developed lumbar backache. He had recently begun to cough and had lost some weight. Examination showed a dense flaccid paraplegia involving all muscles innervated by the 1st sacral and lower segments, with absent deep reflexes in the legs and dense sensory loss below the 1st sacral segment. Plain X-rays of the lumbar spine and sacrum appeared normal. X-ray of chest was considered to show enlargement of hilar glands.

Lumbar puncture gave no evidence of a manometric block; the cerebrospinal fluid contained 120 mgm. of protein per 100 ml. and 18 polymorphonuclear leucocytes and 25 lymphocytes per cubic mm. Myelography was normal. Bronchoscopy was subsequently performed by Mr. J. Howard Brown. A fungating tumour on the lateral wall of the left lower lobe branchus was seen, biopsy of which showed a poorly-differentiated squamous-cell carcinoma. He was then seen in consultation by Dr. J. V. Gordon who considered that the condition was either a subacute necrotic myelopathy or a small secondary deposit in the conus medullaris. He died approximately eight weeks after admission to hospital. Autopsy confirmed the diagnosis of visceral carcinomatosis and necrotic myelopathy of the conus medullaris.

Comment

The clinical picture of a man who is found to have a carcinoma and who presents with a focal spinal cord lesion associated with radicular pain and backache, would readily lead one to suspect spinal cord compression due to metastases. It should be noted carefully, however, that this patient's history differs in one vital particular from the typical history of extradural malignant tumours, in that the first neurological signs appeared simultaneously with the radicular pain and in fact antedated the onset of backache. This may be an important point of differentiation between spinal cord compression by malignant deposits and the myelopathy associated with certain varieties of malignant neoplasm. Myelography in this case clearly excluded spinal cord compression and thus saved a fruitless operation.

OPERATIVE TREATMENT

Little need be said concerning the details of operative technique except to emphasize the objects and principles of operation, namely, to decompress the spinal theca adequately by removal of sufficient laminae and underlying tumour.

The surgeon should be prepared to extend the operation upwards or downwards as necessary until the whole theca is seen to pulsate over the full length of the lesion. This may entail on occasions extensive laminectomies. The most difficult situation is when the tumour mass lies in front of the theca; however, by a more extensive removal of laminae, and perhaps pedicles on one side, the front of the theca can be reached and the tumour removed by small curettes. If necessary one or more nerve root sheaths may be divided after ligation and used to rotate or retract the dural envelope to one side (Perese, 1958). In contrast to the intradural tumours

of the spine, extradural malignant tumours are often extremely vascular and, as many of these patients are in poor nutritional state before operation, blood transfusion is required in nearly every case.

It is unnecessary and undesirable to open the dura at operation unless there is evidence of an intradural component of the tumour. This was so in only one case in this series (Case 1) and similar findings have been noted by other observers (e.g. Rogers, 1958; Perese, 1958).

DISCUSSION AND RESULTS

Even a cursory inspection of this series indicates that, to judge by the standards one would set for the surgical relief of paraplegia in benign spinal tumours, the results are poor. However, when one is faced with malignant disease, and when the object of surgery is purely palliative, one may justifiably demand a less exacting functional result. This is partly a question of one's surgical philosophy. Assessment of results in terms of survival is clearly of little value, except that it indicates faulty surgical judgment to undertake extensive major palliative surgery on an obviously moribund patient who dies within a few days of operation.

The conclusion of Törmä (1957) that, apart from verification of the diagnosis, surgical treatment is useless in cases of malignant spinal tumour because survival times were not improved by surgery, is I submit, based on faulty concepts of the objects of surgical treatment. Few would deny that an ambulant patient with malignant disease is a better proposition than one with paraplegia and malignant disease.

I have grouped these 18 cases into three categories depending on the functional recovery achieved after surgery. Patients who gained sufficient recovery for unaided ambulation have been classified as good results. Those who gained some motor and sensory recovery, insufficient for ambulation, but sufficient to materially simplify nursing procedures by useful mobility in bed were classified as fair results. This group includes two cases (8 and 18) who were discharged from hospital walking unaided after the initial operation. However, recurrence of the paraplegia necessitated their early readmission to hospital and secondary treatment failed to achieve more than a transient functional improvement. Cases in whom there was no useful recovery were classified as poor results. Table 2 indicates that there were exactly a third of the cases in each group. Several possible factors responsible for the good results, or more pertinently the poor results, are worth considering.

TABLE 2

FUNCTIONAL RECOVERY AFTER OPERATION FOR MALIGNANT SPINAL TUMOUR

Good recovery	6 cases (33 per cent.)
Fair recovery	6 cases (33 per cent.)
Poor recovery	6 cases (33 per cent.)

With regard to the type of tumour and the site of the primary lesion, no particular tumour type appeared to carry a more favourable prognosis than any other. There were one or two good results in each of the various groups containing the same type of tumour. It can be concluded, therefore, that type of tumour does not materially influence the functional result of decompression of the cord. In two cases, however, an initial good result was largely nullified by rapid recurrence of the tumour in the extradural space (Case 8, malignant melanoma; Case 17, lymphosarcoma).

The anatomical disposition of the spinal tumour seen at operation may, however, indicate a favourable or a poor prognosis. As mentioned earlier it is rather characteristic of these tumours to encircle the cord like a strangling collar; in the ten patients in whom this was so, the results were poor or only fair in eight cases. In the three patients in whom a circumscribed mass of tumour was found, the results were good. The sinister significance of the collar-like tumour lies in the fact that it not only compresses the cord on all sides but, in addition, it strangles the local and segmental blood supply of the cord producing a localized infarction. This vascular mechanism was presumably predominant in Case 13 (reticulosarcoma) in which only a thin collar of tumour was found at operation without gross compression of the cord.

In considering the length of prodromal symptoms in relation to recovery (Table 3) we find that all the good results occurred in patients who had had local or radicular pain for periods of three months or longer before the onset of cord symptoms, the average period being seven and one-half months. By contrast the patients in whom a poor or only fair result was achieved had a much shorter prodromal period, the average for the two groups combined being three and one-quarter months. This is a manifestation of the rapidity of local growth of the tumour, and is indicative of the more favourable prognosis of slowly growing tumours.

TABLE 3

RECOVERY IN RELATION TO LENGTH OF PRODROMAL SYMPTOMS

Good recovery —	average duration of prodromata — 7½ months.
Fair recovery Poor recovery	average duration of prodromata — 3¼ months.

A comparatively slow progression of cord compression was also noted in the group of good results (Table 4) ranging from periods of eight days to three months from the onset of cord symptoms till operation, and this is in keeping with experience with other forms of cord compression, that is, the slower the development of the compression, the better is the ultimate result.

The neurological status of the patient at the time of operation also influences the prognosis (Table 5). In four cases the cord lesion was absolute at the time of operation. In Case 14 (reticulosis) a fair recovery followed laminectomy but it is probably of considerable importance to note that this man was operated on within 6 hours of his becoming totally paraplegic. In Case 18 (prostatic carcinoma), Case 3 (liposarcoma) and Case 13 (reticulosis) no useful recovery was obtained, all having been totally paraplegic for more than 24 hours before operation. In Case 16 (reticulosis) a complete cauda equina lesion had been present for twenty-four hours before operation and in this case a fair recovery was obtained.

It will be seen from Table 5 that complete motor loss is of evil significance, only one good recovery occurring in this group. This table incidentally illustrates that the motor pathways of the cord are more vulnerable to compression than the sensory pathways, in common with other varieties of cord compression.

In considering these cases as a group, it is clear that decompression at an early stage, preferably before the onset of paralytic signs is the key to a successful result. This implies not only early recognition of the condition, but also early operation after the diagnosis has been made. Perese (1958) rightly regards this condition as a surgical emergency. Given a patient with an incomplete motor and sensory paraplegia from a malignant extra- result following decompression of the cord dural spinal tumour the chances of a good are a little higher than 50 per cent. The presence of complete motor paralysis of more than a few hours duration practically nullifies the chances of a good recovery.

TABLE 4

RECOVERY IN RELATION TO DURATION OF CORD COMPRESSION AT TIME OF OPERATION

Good recovery —	average duration of cord compression — 37 days.
Fair recovery Poor recovery	average duration of cord compression — 4 days.

One's surgical judgment in regard to cases of paraplegia of this type is doubtless influenced to some extent by one's attitude to malignant disease as a whole. Some clinicians feel that evidence of disseminated malignancy calls for only conservative palliation of symptoms, and the evidence presented here indicates that when complete paraplegia has been allowed to develop surgery has little or nothing to offer. Whilst no one would claim that the surgery of malignant spinal tumours is a particularly gratifying experience, the results obtained in incomplete paraplegia satisfy the limited aim of surgery in these cases. Sequential use of improved methods of palliation in other directions, such as radiotherapy,

chemotherapy and endocrine ablation for hormonal dependent cancers, may well result in further control and useful survival.

Operative treatment should consist of laminectomy and decompression of the spinal dura by removal of sufficient tumour from the extradural space to restore pulsation to the compressed segment of dura.

TABLE 5

RECOVERY IN RELATION TO NEUROLOGICAL STATUS AT TIME OF OPERATION

	Good	*Fair*	*Poor*
Absolute paraplegia	–	2	3
Complete motor paralysis and partial sensory loss	1	–	3
Partial motor paralysis and partial sensory loss	5	4	–
Partial motor paralysis and complete sensory loss	No cases		

SUMMARY

Some aspects of the pathology of malignant spinal tumours is discussed and the importance of the vertebral venous system in determining the mode of dissemination of these tumours into the spinal intradural space is emphasized. The rarity of spinal intradural metastatic tumours is noted and possible reasons are given. The intradural spinal veins probably do not participate in the shunting function of the vertebral venous system *vis-à-vis* the caval venous system.

The clinical features of 18 cases of malignant extradural spinal tumour are presented. The diagnostic importance of the prodromal stage consisting of either local spinal pain or of root pain is stressed. Whereas prodromal symptoms are often prolonged, cord compression often develops rapidly and opportunities for successful treatment diminish rapidly with the onset of paralysis. Diagnosis can be confirmed in all cases by radiographs of the spine, including myelography if necessary.

Good functional results (unaided ambulation) were achieved in one-third of the cases. The important factors in achieving a satisfactory result are early diagnosis and operation, if possible, before the onset of paralytic signs. Once motor paralysis is complete surgical treatment is almost valueless.

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ACCESSORY URETHRA IN THE MALE CHILD*

REPORT OF TWO CASES

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ACCESSORY urethra is a rarely encountered anomaly and the purpose of this paper is to place two cases on record. One belonged to the rarer group of complete accessory urethra, its distinguishing feature being that the abnormal channel was preferentially used; the other represented a more extreme form of the commoner incomplete group.

There have been many and varied classifications of accessory urethra; that of Gross and Moore (1950) which is adopted in this report, has two main groups.

1. Complete accessory urethra

Here there is an accessory channel communicating from the bladder to the exterior. The track is always dorsal to the normally placed urethra and it may open into the exterior at the glans penis, the coronal sulcus, the base of the penis or on the dorsal surface of the penile shaft. If the external opening is not on the glans, there is usually a dorsal epispadial groove and there may be a dorsal chordee.

2. Incomplete accessory urethra

This group comprises those accessory channels which do not open into the bladder. They lie either dorsal or ventral to the normal urethra and extending for variable distances, may be blind or may communicate with the normal urethra.

There have been scattered reports of accessory urethra and these were summarized by Gross and Moore in 1950. Of the 83 cases of accessory urethra then on record, 19 were complete accessory urethrae. Since then Arnold and Kaylor (1953), Slotkin and Mercer (1953), Funfack (1953), Mason Brown (1956) and Wrenn and Michie (1957) have reported 5 more examples of the complete urethrae. Ravitch (1953) reported a patient in whom the duplicate urethra was part of a larger anomaly involving duplication of the bladder and large bowel.

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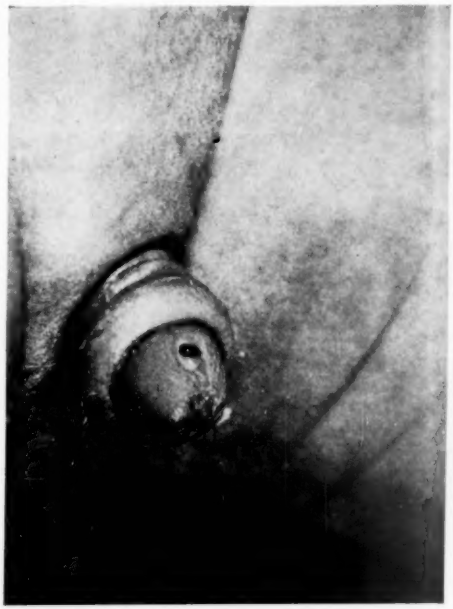

FIG. I. Photograph of penis in Case 1 to show both the accessory urethral orifice and the normal urethral meatus on the glans.

Case 1. Complete accessory urethra

M.D., male, aged 1 year and 10 months, was admitted to the Royal Children's Hospital on 29th January, 1958.

His mother had noticed that he passed urine through either or both of two orifices of the glans penis. The dorsal of these two orifices was preferred. The child was well developed and could control micturition. There was no history or evidence of infection of the urinary tract. There were no relevant details in the family or past histories.

The child was healthy. Two orifices were noted on the glans penis. One of these was normal in appearance, calibre and situation. The other was a smaller stenotic orifice, approximately 0.2 cm. in diameter and situated in the midline on the dorsum of the glans distal to the corona (Fig. I). The external appearance of the shaft of the penis was normal. There was no dorsal chordee or groove or abnormality of the prepuce.

Intravenous pyelogram showed a normal right calyceal and ureteric system. No excretion of dye from the left side was seen and there was no discernible renal shadow.

Two urethrae were demonstrated by urethrography, the dorsal one emerging from the bladder, a little anterior to the other (Fig. II). Both urethrae ran a parallel course. The accessory urethra was the smaller in diameter.

FIG. II. Urethrograms showing the normal and accessory urethrae of Case 1.

Cystoscopy and urethroscopy performed through the ventral urethra revealed a normal posterior urethra and verumontanum. The anterior wall of the bladder exhibited a V-shaped notch but the accessory urethral orifice was not visible. The right ureteric orifice was normal in position. No ureteric orifice was seen on the left side. Five days before operation the dorsal glandular orifice was closed by suture to confirm the functional adequacy of the ventral channel. A good controlled stream from the normally situated urethra was demonstrated on the following days.

At operation on 7th February, 1958, the accessory urethra was exposed both in the retro-pubic and in the penile segments of its course. The posterior part of the accessory urethra was displayed in the retro-pubic region. It was incorporated in the anterior wall of the orthotopic urethra and was dissected from its orifice in the bladder down to the region of the pubic arch. Through a midline incision the bladder was opened and the orifice of the accessory urethra was seen to be approximately 1.0 cm. anterior to the opening of the normal urethra.

Through a short midline incision on the dorsal aspect of the penile shaft, the accessory urethra was dissected proximally and distally until the whole tract was excised. The distal urethra was tunnelled in the subcutaneous plane to the glans where it was divided at the orifice.

The bladder was drained by an indwelling supra-pubic catheter and both incisions were closed in layers.

Post-operative convalescence was uneventful. He was subsequently continent of urine and could pass a good stream through the normal urethra.

Case 2. Incomplete accessory urethra

G.A., aged 1 year and 8 months, was admitted on 28th August, 1960.

At the age of six weeks two orifices on the glans penis were noted by the practitioner who was consulted for advice on circumcision. Urine emerged only from the ventral normally placed opening.

There was no history of infection, either locally or in the urinary tract. The family and past history revealed nothing relevant. The boy was healthy and exhibited no other abnormalities.

Two orifices were seen on the glans. One of them was normal in appearance and situation. The other was situated on the dorsum of the glans distal to the corona. No dorsal chordee or groove of the penile shaft was present.

A cysto-urethrogram revealed a normal ventral urethra. Dye introduced into the other orifice outlined a narrower dorsal tract parallel to and above the normal urethra. It petered out at a point contiguous with the base of the bladder in the region of the bladder neck. There was no demonstrable communication between this channel and the bladder or urethra.

At operation on 29th August the urethra was approached by retro-pubic and penile incisions. A lower midline abdominal incision was made and the bladder neck and retro-pubic region were explored. No track was seen. The bladder was opened and no accessory orifice was visible. Two ureteric orifices and the normal internal meatus were identified.

A midline incision near the base of the penile shaft was made. The termination of the tract in the glans was cored out and the tract was then dissected from the shaft of the penis towards the bladder. This led in the midline to a point a few millimetres cranial to the perineal membrane where the lumen of the track terminated in a strand of fibrous tissue. The tract was divided at this point.

The bladder was drained by an in-dwelling urethral catheter and the incisions were sutured in layers. The child was discharged after ten days, voiding satisfactorily through the normal urethra.

COMMENT AND DISCUSSION

The first of these two cases falls in the rare group of complete accessory urethra. At the present time the number of reported cases is less than thirty. In the patient reported here, the left kidney may be presumed to be absent. This combination has not been recorded hitherto. Another noteworthy feature was the more frequent passage of urine down the narrower and stenotic accessory urethra, which appeared to be under efficient control of a sphincter mechanism.

Furthermore, after excision of the accessory urethra, the normal urethra functioned normally and has done so for the last two and one-half years, since operation.

The second patient illustrates the commoner group of incomplete blind accessory urethra. The channel terminated as a strand of fibrous tissue near the bladder neck. In this group the accessory channels are not usually as long as that in the child G.A. This case therefore represents a more extensive developmental aberration of the incomplete type of accessory urethra.

ACKNOWLEDGEMENT

These patients were studied whilst I was attending the Royal Children's Hospital for a short period as supernumerary registrar.

I am grateful to Mr. F. Douglas Stephens and to Mr. R. Fowler, Jnr., for permission to assist at operation on Case 2 and to use the clinical records in the preparation of this paper.

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THE COURSE OF PROCTITIS AND MILD PROCTOCOLITIS*

By E. S. R. Hughes

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THERE is little information available concerning the course of patients suffering from proctitis or from proctocolitis involving part of the left half of the colon with minimal or no radiological changes. With this in mind, a personal series of cases treated three or more years ago was reviewed. These patients had been indexed, progressively reviewed and contact had been maintained with them.

Cases in Series

There were 74 cases treated three or more years ago and all but 5 were available for study. There were 48 females and 26 males. The average age in both sexes at the time of consultation was 39 years. The average age of onset was 36 years.

Patients complained of bleeding and usually some frequency of bowel action or incontinence on passing flatus. Sigmoidoscopy revealed an oedematous, red and friable mucous membrane and a variable amount of exudate in the lumen.

If the inflammatory changes were confined to the rectum with normal mucous membrane observed in the upper rectum or sigmoid colon, the case was classified as proctitis. There were 47 such cases. A barium enema was not considered necessary in this group.

If the whole rectum and recto-sigmoid colon were involved and it was not possible to reach normal mucous membrane at sigmoidoscopy, the case was classified as proctocolitis. There were 27 cases with mild proctocolitis. A barium enema in this group was sometimes normal (9 of 23 X-rays reviewed), sometimes showed minimal changes in the sigmoid and perhaps descending colon (11 of 23 cases) and sometimes was equivocal (3 of 23 cases).

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Treatment

After establishing the diagnosis, the nature of the trouble was explained to the patient and a reassurance given that there was no cancer present. Hospital admission immediately after first consultation was advised in 2 patients from country districts because of convenience, but the remainder were treated as Out-patients. A low- or non-residual diet was advised, sometimes combined with methylcellulose. Codeine, phenobarbitone or belladonna proved helpful on occasions. Chemotherapy and antibiotics were used rarely. Steroids were not given in this series.

Results

Of the 47 patients with proctitis, records were complete in 44. Thirty-five (80 per cent.) were well and more or less free of symptoms but it was the rule to find some evidence of proctitis on sigmoidoscopy. Four patients had intermittent exacerbations, but they were not troublesome. In 5, the inflammation extended into the colon and 2 (5 per cent.) required surgical treatment (Table 1).

Of the 27 patients with proctocolitis, 25 were traced. Thirteen (52 per cent.) were untroubled by the disease, although sigmoidoscopy in each showed that it persisted, often mildly. Seven patients (28 per cent.) continued to have intermittent symptoms. Four patients (16 per cent.) required surgery and a fifth was having a troublesome time (Table 1).

Discussion

It is accepted by most that proctitis is a localized form of ulcerative colitis (Hughes, 1956; Newell and Avery Jones, 1958). One such patient, Mrs. P., aged 32 years, at the first consultation in April, 1955, had noticed bleeding and some diarrhoea for three years. Examination showed proctitis to 9 centimetres. She was examined at six-monthly

intervals and the appearances remained unchanged until April, 1959, when a severe exacerbation was found to be associated with extension into the sigmoid colon. A barium enema was normal. Within a few weeks there was a spontaneous remission and sigmoidoscopy showed a return to the original proctitis.

three times daily for two or three weeks, gradually tapering off). Five had no improvement and 11 had persistent symptoms and signs. This gives an overall unsatisfactory result of 16 (36 per cent.).

Six of the 69 patients studied in this series (8.7 per cent.) required surgical treatment.

TABLE 1

THE 6 PATIENTS WHO PROGRESSED TO SURGICAL TREATMENT

No.	*Name*	*Sex*	*Age*	*Duration disease*	*Date of consultation*	*Classification*	*Complication*	*Date of operation*
1	B	M	42	3 months	August, 1954	Proctitis	Toxaemia	March, 1958
2	W	F	62	1 month	April, 1955	Proctitis	Toxaemia Distension	May, 1955
3	K	M	30	3 years	May, 1956	Proctocolitis	Toxaemia Perforation	August, 1956
4	McG	M	63	5 years	September, 1956	Proctocolitis	Toxaemia	January, 1957
5	H	F	38	1 year	May, 1957	Proctocolitis	Chronic diarrhoea	December, 1959
6	W	F	18	1 month	June, 1957	Proctocolitis	Toxaemia	April, 1958

In this series, 48 of the 69 patients traced were well and relatively free from symptoms three years or more after the initial consultation but it was the rule to find sigmoidoscopic evidence of persistent disease. In 21 (30 per cent.) symptoms continued, although not necessarily to a troublesome degree. In a series of 144 cases reviewed by Newell and Avery Jones (1958), 50 (35 per cent.) had continuing symptoms. These authors do not analyse their figures further in this communication, but in the writer's series, 9 of 44 patients with proctitis (20 per cent.) and 12 of 27 patients with proctocolitis (44 per cent.) continue to have symptoms. Kiefer and Jordan (1946) reported 97 cases of proctitis and partial colitis and 37 (38 per cent.) had a poor result. Nine (26 per cent.) of 34 patients with proctitis followed for two years or more had a poor result and 28 (44 per cent.) of 63 patients with partial colitis.

Newell and Avery Jones (1958) treated 44 of their cases with prednisone (20 milligrams three times a day for a week; 15 milligrams three times daily for a week; 10 milligrams

Two of the 6 cases had proctitis initially but both developed very severe generalized extension of the disease and died despite surgery. The other 4 cases had proctocolitis when first seen; in 3 of these acute toxaemic ulcerative colitis supervened and in the fourth the disease was responsible for persistent diarrhoea incompatible with comfortable existence. All four survived surgery. Operation was required in 12 of the 144 cases (8.3 per cent.) studied by Newell and Avery Jones (1958). Truelove and Witts (1959) in reporting the results of cortisone and corticotrophin in ulcerative colitis state that 14 of the 169 patients (8.3 per cent.) required surgery during or shortly after the trial period.

Zetzel and Atin (1958) in their review of the value of ACTH and adrenalcorticosteroids in the treatment of ulcerative colitis emphasize the suppressive rather than the curative effects, stating that, "The high incidence of relapses even on maintenance therapy enhances the need for continuous medical supervision . . ."

The course followed by these cases of proctitis and proctocolitis in the writer's series compared with those treated by steroids suggests that this latter therapy has little overall effect on the disease.

CONCLUSIONS

Proctitis is a localized form of ulcerative colitis and progresses satisfactorily without special treatment in 80 per cent. of cases. In about 10 per cent. of cases symptoms persist and cause occasional annoyance to the patient. In another 10 per cent. the disease extends into the colon and about half of these will require surgery.

As might be expected, the more extensive proctocolitis with or without radiological changes has not got quite the same good prognosis. About 50 per cent. of these patients progress well. Approximately 30 per cent. have intermittent mild exacerbations which prove troublesome from time to time. About 20 per cent. require surgery.

There is still no satisfactory evidence to show that steroid therapy alters the course of these mild forms of ulcerative colitis.

SUMMARY

Seventy-four cases of proctitis and mild proctocolitis treated three or more years ago are reviewed. There were 48 females and 26 males. The average age in both sex groups was 39 years. There were 47 patients with proctitis and 27 with proctocolitis. No "specific" treatment was given to this group of patients. Of the 47 patients with proctitis, 44 were followed for three years or more and were re-traced. Thirty-five (80 per cent.) were relatively free from symptoms. Four (9 per cent.) had intermittent excerbations without extension of the disease. Five (11 per cent.) had extension of the disease and 2 of these required surgery. Of the 27 patients with proctocolitis, 25 were available for the three year or more study. Thirteen (56 per cent.) were relatively free from trouble. Seven (28 per cent.) continued to have intermittent symptoms. Four patients (16 per cent.) came to surgery and a fifth appeared likely to do so. The few published results of steroid therapy do not appear to show any improvement on these results.

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HAEMORRHAGIC INFARCTION OF THE TESTIS IN THE NEWBORN*

By H. D. D. Tyer

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HAEMORRHAGIC infarction of the testis is far more frequent in the newborn than is generally recognized. The condition may arise spontaneously or may follow testicular torsion. Cases overlooked at birth are probably responsible for many atrophic testes discovered in later life.

Historical Review

Torsion of the testis at birth was first described by Taylor (1897) and only three more reports appeared in the English literature over the next half-century (Begg, 1921; Campbell, 1948). During the past decade an additional 18 cases of proven torsion of the testis in the newborn have been recorded (Biorn *et alii*, 1951; James, 1953; Longino *et alii*, 1955; Franzblau, 1956; Tankin *et alii*, 1958).

Infarction of the testis in the absence of demonstrable torsion has been described on 11 occasions. Some authors have postulated the occurrence of spontaneous detorsion prior to surgery in such cases (Nash, 1921; Rhyne *et alii*, 1955, Thiel, 1956; Pinto *et alii*, 1957). Others have denied this possibility and regarded the condition as idiopathic infarction of the testis (Maclean, 1943; Ravich, 1947; Fernicola, 1954; Tankin *et alii*, 1958; Hogan, 1959).

A sharp line of demarcation of the infarct at the lower portion of the spermatic cord has been advanced as evidence in favour of previous torsion (Rhyne *et alii*, 1955). In 3 cases this line has been associated with a fibrous band or constriction (Maclean, 1943; Pinto *et alii*, 1957). Spontaneous thrombosis of the testicular artery has been found on 2 occasions (Ravich, 1947; Hogan, 1959).

Birth trauma appears to play no part in the aetiology of this condition. No case presented as a breech and 2 were delivered by Caesarean section (Biorn *et alii*, 1951; Rhyne *et alii*, 1955).

Several authors have found histological features suggesting long-standing infarction. Biorn (1951) described fibrous adhesions in an infant one day old. Ravich (1947) and James (1953) discovered areas of calcification in the testes of infants undergoing surgery on the sixteenth and twenty-first days of life respectively.

A scrotal mass was detected within a short time of delivery in most cases. On only 3 occasions was the initial discovery made later than the first day of life (Nash, 1921; Tankin *et alii* 1958).

Despite this early detection, only one case was explored within twenty-four hours of birth (Franzblau, 1956). The average time of surgery was the eighth day after delivery, and in 6 cases the delay exceeded two weeks.

A correct diagnosis was infrequent. Many cases came to surgery at a late stage with a mistaken diagnosis of testicular neoplasm when the mass failed to resolve under observation. In no case has the testis appeared viable at the time of operation.

Of the 22 cases with proven torsion of the testis, only 3 appeared to have any of the anatomical abnormalities said to predispose to torsion of the testis in adults (Taylor, 1897; Begg, 1921; Franzblau, 1956). No case has been recorded in a mal-descended testis. More than half of those with torsion were of the rare extravaginal type, the twist being located between the superficial inguinal ring and the upper pole of the testis, above the tunical sac (Campbell, 1948; Longino *et alii*, 1955).

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Orchidectomy has been performed by all authors except Longino and Martin (1955). They performed simple detorsion in the last 7 of their nine cases, regardless of the necrotic appearance of the testis. No complications arose from this procedure. In 2 cases subsequent atrophy seemed minimal despite delays of four and five days in performing detorsion. No biopsies were taken at later stages to confirm the presence of functional testicular substance.

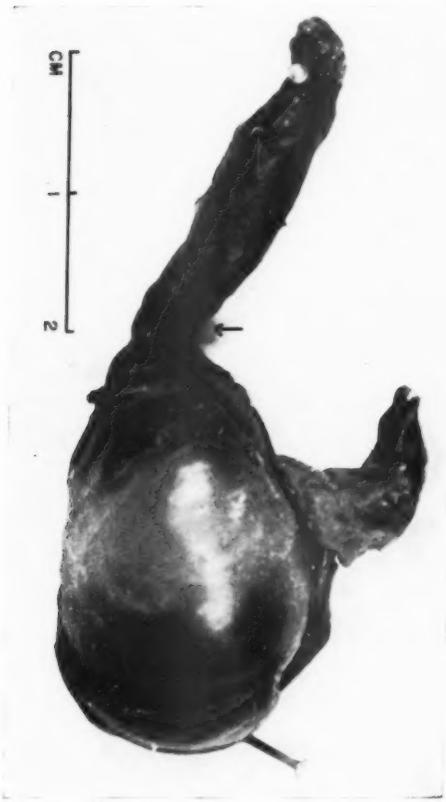

FIG. I. Operative specimen from Case 1. The site of torsion is marked by the arrow at the lower end of the spermatic cord. Beyond this point all tissues are infarcted including both layers of the tunica vaginalis. A portion of the parietal layer of the tunical sac is seen on the right border of the testis.

CASE REPORTS

Case 1

Baby H was delivered by Caesarean section following the onset of signs of foetal distress during the first stage of labour. The right side of the scrotum was seen to be swollen at delivery.

Subsequent examination revealed an apparently tender mass in the right hemiscrotum twice the size of a normal testis. The cord was thicker than on the left and the skin overlying the mass was red and oedematous.

A provisional diagnosis of testicular torsion was made and the scrotum was explored fourteen hours after birth. The tunica vaginalis was purple and contained clotted blood. The testis and epididymis were swollen and almost black in colour (Fig. I). No developmental abnormality was detected.

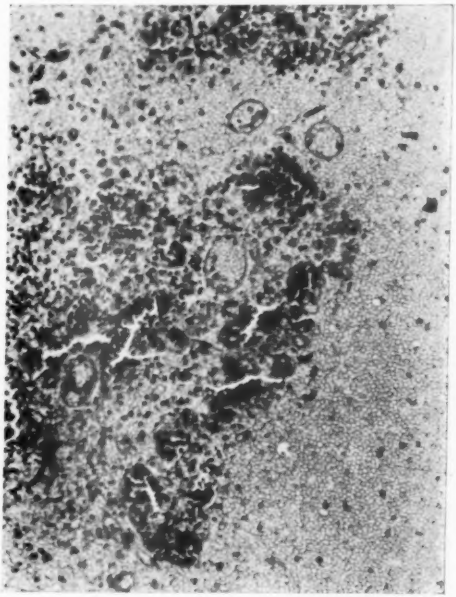

FIG. II. High power photomicrograph from Case 1. There is a diffuse haemorrhagic infarction of the testis. Much of the normal testicular tissue is disrupted by sheets of extravasated red blood cells.

Further inspection revealed a twist of the spermatic cord above the level of the tunical reflection leading to an extravaginal torsion of the testis and tunical sac.

Detorsion was attempted without success and orchidectomy was then performed. The specimen measures 3 x 2 cm., its cut surface having the appearance of an old blood clot.

Histological examination revealed a diffuse haemorrhagic infarction of the testis, epididymis and tunical sac. Large areas of testicular tissue were replaced by sheets of red blood cells. As most of these were still intact and no iron pigment could be demonstrated with Prussian blue stains, it was felt that infarction was of fairly recent onset (Fig. II).

Case 2

Baby C was found to have a swollen scrotum after a normal vertex delivery.

A mass 3 times the size of a normal testis could be felt in the left side of the scrotum, associated with a low-tension hydrocoele. Pressure evoked obvious signs of tenderness. The cord was thickened and the skin over the mass was purple. A diagnosis of testicular torsion was made.

The scrotum was explored ninety minutes after birth. The tunical sac was mildly congested and contained a small volume of sero-sanguinous fluid. The testis and epididymis were quite gangrenous.

There was a high reflection of the tunica vaginalis on to the lower portion of the spermatic cord. The testis and epididymis were freely suspended within the tunical sac without any sign of a mesorchium.

The intravaginal portion of the cord was the site of a double twist which produced such constriction that spontaneous separation seemed likely.

Orchidectomy was performed. The testis measured 3.5 x 2.5 cm and examination revealed diffuse haemorrhagic infarction.

DISCUSSION

All testicular swellings discovered in newborn infants should be diagnosed as haemorrhagic infarction of the testis. It is not possible to predict the presence or absence of an associated torsion without surgical exploration.

The mass may be 2 to 4 times the size of a normal testis and is rarely tender. The overlying skin is almost invariably discoloured blue, or red and may be oedematous. The cord is thickened in only a minority and may be found in the absence of torsion.

Adherence to the scrotum occurs if exploration is delayed but abscess formation has only been reported following attempts at aspiration (Campbell, 1948). Signs of systemic toxicity are absent unless infection has supervened.

An associated hydrocoele is commonly present and occurs on the normal and pathological sides with equal frequency.

Fernicola (1954) has stressed the difficulty of excluding a neoplasm without exploration. Such a finding however appears unlikely. In an extensive survey of congenital malignancies, Wells (1940) could find only 4 authentic cases involving the testis. Since then Melicow (1955) has added a case of embryonal carcinoma in a neonate.

Benign tumours of the testis (Fam *et alii*, 1958) and epididymis (Burros *et alii*, 1950) are equally rare in the newborn, while James (1953) considers that acute epididymo-orchitis is so rare in infancy that such a diagnosis should never be made.

The high incidence of the rare extravaginal type of torsion in these cases is remarkable. It contrasts with the pathology of the adult condition where torsion is usually associated with some developmental abnormality of the testis (Donovan, 1930; Wheeler, 1952; Woodson, 1958) and is almost invariably of the intravaginal type (Ewart *et alii*, 1944; Lovell, 1950).

This difference appears to arise from the tenuous attachments of the testis and tunical sac to the scrotum of the foetus or newborn infant (Hunter, 1926). Rotation of the entire contents of the hemiscrotum can thus occur in a manner impossible in later life.

Early diagnosis and operation are widely accepted as essential points in the management of torsion of the testis. When the condition arises *in utero* however, the scales weigh heavily against the surgeon. Irreparable damage is likely by the time of delivery and the incidence of successful salvage must necessarily be very low.

Despite this air of pessimism, operation is still justifiable if the scrotal mass is detected at birth. It is dubious if exploration should be carried out after the first day of life. There appears to be no risk involved in leaving the testis *in situ* even when it appears gangrenous. The possibility that some residual endocrine function remains in these infarcted testes cannot be excluded.

Exploration of the normal side is only warranted in the small percentage of cases in which anatomical abnormalities are detected at operative detorsion. That such abnormalities may be bilateral is shown by a case described by Franzblau (1956) where a prophylactic orchidopexy was performed on the non-infarcted side for a "clapper bell" deformity.

SUMMARY

1. Haemorrhagic infarction of the testis in the newborn may arise spontaneously or follow testicular torsion.

2. Intra-uterine torsion of the testis differs from the similar condition in later life. The testis is usually normal in development and the torsion is frequently extravaginal in type.
3. Detorsion at birth offers the only chance of salvaging testicular function. No ill effects occur if the infarcted organ is left *in situ* after detorsion.
4. Two cases of intra-uterine torsion of the testis are presented, one being extravaginal and the other intravaginal in type. They are the second and third cases reported in the English literature to have surgery for this condition on the first day of life.

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THE ELECTROENCEPHALOGRAM IN CARDIAC SURGERY*

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"Knowledge is of two kinds. We know a subject ourselves,
or we know where we can find information upon it."
—Dr. Samuel Johnson.

THE use of the electroencephalogram as an aid in surgery is becoming more prevalent each year. This paper is designed to present a broad survey of some of the relevant literature combined with personal experience in the Thoracic Surgical Unit at the Alfred Hospital, Melbourne.

Methods

An Edin Anesthograph† is used which combines facilities for the recording of electroencephalograms and electrocardiograms. The electroencephalogram produces a single channel record by the use of stainless steel wire electrodes which are placed subcutaneously for fronto-occipital derivation as suggested by Courtin, Bickford and Faulconer (1950). After induction of anaesthesia the frontal electrode is placed on the frontal eminence one inch from the midline. The occipital electrode is positioned in direct line with the frontal lead one and a half inches from the external occipital protuberance. A third electrode is placed over the mastoid process to act as an earth.

The speed of the paper is 15 mm. per second. However, as international speed is 30 mm. per second the machine has recently been modified to this standard. The standard amplitude deflexion is 100 microvolts equals 5 mm.

In an endeavour to reduce interference from other electrical equipment in the theatre the frequency response of the electroencephalographic channel is limited to between 1 and 30 cycles per second. At 0.75 cycles per second and 30 cycles per second the response is reduced to 50 per cent. At 0.3 cycles per second and 50 cycles per second the response is zero.

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†Instrument made by Edin Instrument Co. Inc.

Artefacts which can interfere with the recording are caused by movement of the patient or cables, one electrode not connected or inefficient earthing of the machine.

Discussion

Genesis of the electroencephalogram

The origin of the electrical oscillations of the electroencephalogram is still unknown. Wyke (1955, 1957) has suggested that the electroencephalogram represents the activity of a patient's cortical neurones and that these neurones are driven into action by the continuous flow upwards of impulses from the underlying reticular nuclei of the brain stem.

The position of the lateral reticular formation is in the brain stem extending from the lower limits of the fourth ventricle to the hypothalamus. This complex receives sensory information from spinal and cranial nerves. The cortex receives impulses from the well recognized long tract systems and also from the lateral reticular formation.

Any factors which influence the excitability of cortical neurones or the discharge from the reticular nuclei, or both, will produce similar changes in the electroencephalogram and the patient's conscious state; the changes depending upon the strength and nature of these applied factors.

Whereas Wyke interprets the electroencephalogram as activity from the cortical neurones, Li and Jasper (1953) consider that the electroencephalogram is derived from synaptic potentials and not action potentials of the cortical cells. These workers have shown in cats that unitary spike discharge from cortical cells can be modified without affecting the electroencephalogram by barbiturate anaesthesia or convulsants. The presumption is that transmission across the

synapses deeper in the brain must occur before a cortical cell can fire and that the transmission across the synapse is prevented by anaesthesia before the synaptic potential is reduced.

Principles of use of the electroencephalogram

The electroencephalogram obtained for use in anaesthesia differs in one major aspect from that used in neurology. In anaesthesia it is the overall response of the cortex which is desired, not a localization of a particular focus. Therefore, as shown by Courtin, Bickford and Faulconer (1950), fronto-occipital derivation is adequate and multi-channel recording is not needed.

Pearcy and Virtue (1960) have shown that with multi-channel recording during hypothermia and circulatory occlusion that the occipital areas tended to develop changes one to three seconds before the frontal regions.

Analysis of the waves of the electroencephalogram is done under two major divisions, the frequency in cycles per second and the amplitude in microvolts. The general configuration of the complex is of minor interest.

There is not much electrical activity from the brain in the first three months of life. Activity is then noted at about 3-4 cycles per second and the frequency gradually advances with the establishment of normal adult rhythm by 8-10 years of age .

Classically the frequency spectrum is divided into five main groups varying between 1-30 cycles per second.

1 – 3.5	c.p.s.	delta
4 – 7	„	theta
8 – 13	„	alpha
14 – 18	„	beta
20 – 30	„	gamma.

A conscious but resting record has a frequency of 8-10 cycles per second with an amplitude of about 50 microvolts. Any visual or mental activity will desynchronize this basic activity. Under the effects of light anaesthesia the frequency is about 10-15 cycles per second and an amplitude of approximately 30-40 microvolts.

The electroencephalogram can be changed by many factors. The effects of anaesthetic agents have been investigated by Courtin, Bickford and Faulconer (1950) for nitrous oxide, oxygen and ether; Kiersey, Bickford and Faulconer (1951) for thiopentone; Possati, Faulconer, Bickford and Hunter (1953) for cyclopropane; Gain *et alii* (1957) for halothane. Gibbs *et alii* (1937) investigated the effect of several drugs on the electroencephalogram; Clowes, Kretchmer, McBurney and Simene (1953) discussed the use of the electroencephalogram in the evaluation of the effects of anaesthetic agents and carbon dioxide accumulation during surgery; Holmberg (1953) investigated the electroencephalographic changes during hypoxia and hyperventilation.

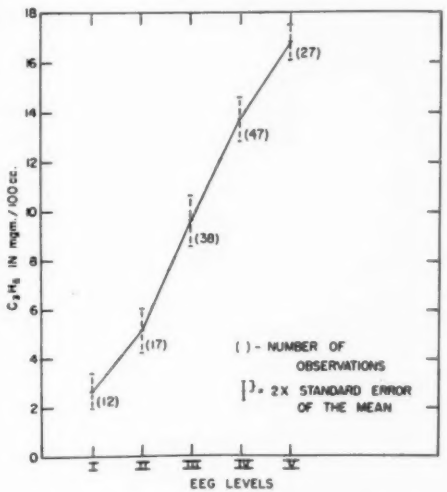

FIG. I. Correlation of arterial level of cyclopropane with the electroencephalogram level. (After Possati *et alii*.)

Faulconer (1952) with ether and Possati *et alii* (1953) with cyclopropane showed that the concentration of these agents in the blood compared with the electroencephalographic changes resulted in a straight line relationship.

Fig. I shows the value of the electroencephalogram in a quantitative assessment of at least one consistent effect of the drug.

The changes in the electroencephalogram associated with deepening anaesthesia have been classified for several of the common anaesthetic agents.

Fig. II shows the changes occurring with progressively deeper halothane anaesthesia.

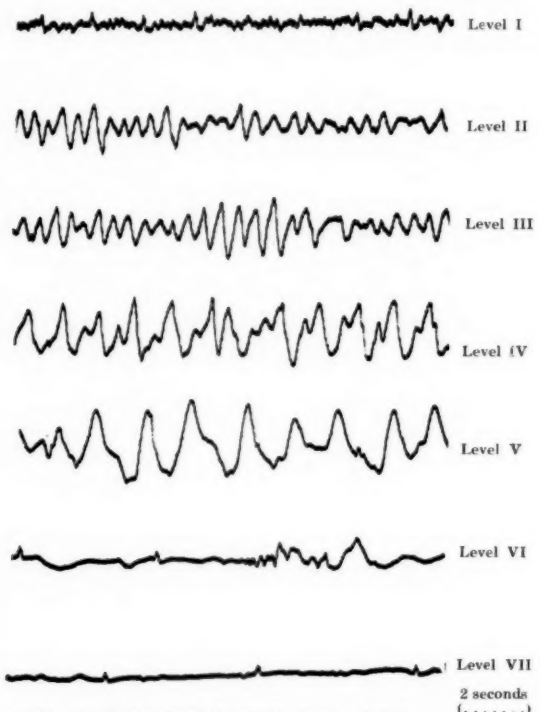

FIG. II. Electroencephalographic levels of halothane anaesthesia. Note the change from fast activity in level I to complete cortical suppression in level VII. Level III gives good operating conditions. Below this level there is severe physiological disturbance.

This classification could be used for any of the common anaesthetic agents providing it is realized that any variations are probably due to different rates of induction rather than any two anaesthetic agents producing dissimilar patterns.

Galla, Rocco and Vandam (1958) found that the correlation of electroencephalogram changes with the classical signs of anaesthetic depth, such as pupil size, muscle tonus, respiratory pattern, etc., was difficult to establish.

The electroencephalogram can be changed in a retrograde fashion by several means. The dose of the drug can be increased or another agent can be added, e.g. nitrous oxide, oxygen and ether or the combination of hypothermia and circulatory occlusion. It has been shown by Faulconer (1952) that a concentration of nitrous oxide in excess of 10 milligrams per 100 millilitres of blood reduced the amount of ether needed to reach a given electroencephalographic level of anaesthesia. A combination of hypothermia and hypoxia produces a deeper electroencephalographic level than either factor alone.

With the use of the electroencephalogram in cardiac surgery it is assumed that the level of anaesthesia is constant and light, and therefore any changes produced in the

electroencephalogram are due to alterations in the cerebral perfusion or cerebral oxygenation.

The major causes of these changes are:

1. Hypotension.
2. Superior vena caval obstruction.
3. High venous pressure.
4. Cerebral vasoconstriction.
5. Hypoxia.
6. Hypothermia.

At the Alfred Hospital changes have been classified differently and a comparison of these two is shown in Table 1.

When by-pass circulation is commenced there is always a retrogression of the electroencephalogram unless, because of the patient's general condition, the machine can provide a better perfusion than the patient.

The reason for this series of changes is not proven. Originally, it was thought to be due to a lowering of the patient's carbon dioxide as 100 per cent. oxygen was supplied to the

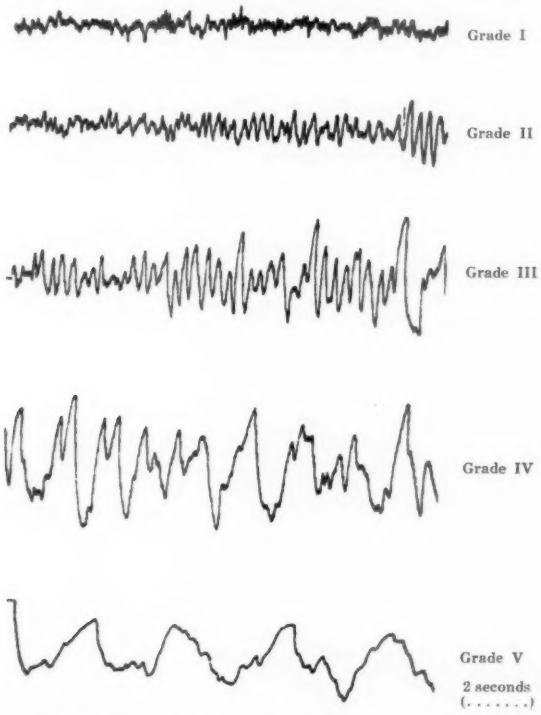

FIG. III. Electroencephalographic grades of anaesthesia under cardiac-pulmonary by-pass.

Fig. III illustrates the changes produced in the electroencephalogram during cardiac by-pass due to rapid lowering of blood pressure.

Hudson (1958) has suggested a table of electroencephalographic levels during cerebral perfusion. However, this was constructed as a result of experimental work with dogs.

oxygenator. However, the change has persisted even in the presence of 5 per cent. carbon dioxide and 95 per cent. oxygen.

Theye, Patrick and Kirklin (1957) suggested that it was due to the difference in temperature between the blood of the machine and that of the patient. However, at the

Alfred Hospital the machine blood is brought to the same temperature as the patient prior to by-pass.

The explanation now appears to centre round the drop in blood pressure which nearly always occurs as by-pass is commenced.

by 40-60 mm. of mercury within the first twenty to thirty seconds.

Several authors have found that drug induced hypotension has been accompanied by maintenance of cerebral blood flow, probably due to decreased cerebro-vascular resistance. Dewar (1953) showed that 30 per cent. de-

TABLE 1

Grade	*Frequency in c.p.s.*		*Amplitude in microvolts*	
	Hudson	*Alfred Hospital*	*Hudson*	*Alfred Hospital*
I	22	18	25	25
II	16	8	30	50
III	5	5	50	150–200
IV	1.3	1 3–4	50	150 50
V	1.3	1–2	10	150
VI	–	–	–	–

Hypotension

It has been shown by several workers that hypotension produces changes in the electroencephalogram. However, the important piece of work was done by Schallek and Walz (1953). In their work it is shown that it was not so much the actual level of blood pressure but the rate of fall of the blood pressure. They have been able to lower the blood pressure of dogs to 40 mm. of mercury without demonstrable changes in the electroencephalogram. However, reducing the blood pressure from 130 to 100 in 30 seconds, i.e. 60 mm. of mercury per minute profoundly depressed the electroencephalogram. Recovery of the trace then occurerd even though the blood pressure was lowered to 60 mm. of mercury. In fact, recovery was almost back to normal but this second depression of blood pressure was taken quite slowly. These workers concluded, therefore, that provided the blood pressure is lowered at a rate not faster than 10 mm. of mercury per minute it is possible to lower the blood pressure of the dog by "Arfonad" to 40 mm. of mercury and hold it at this level for one hour without changing the electroencephalogram.

It has been the experience at the Alfred Hospital that with the commencement of by-pass the blood pressure nearly always drops

pression of blood pressure by hexamethonium had little or no effect on cerebral blood flow (mean blood pressure from 153 to 107). However, Morris (1953) found with a 40 per cent. reduction there was a significant reduction in cerebral blood flow but in this study the mean blood pressure was reduced from 104 to 62.

Therefore it appears that three factors are important in assessing the effects of hypotension, the percentage reduction and actual level of the blood pressure, and of more importance, the rate of reduction of the blood pressure.

Superior vena caval obstruction

When the superior vena caval cannula is placed in position it is intended to be a drain for the venous blood from the brain. However, this can be interrupted by kinking of the cannula, mechanical displacement of the heart or if the loop placed round the superior vena caval cannula is pulled up too tightly. This is one of the important causes of a poor electroencephalogram.

Theye, Patrick and Kirklin showed that in 13 of 100 patients they had retrogression of electroencephalogram on cannulation of superior vena cava or surgical compression

of it. Our experience has shown the complication to be present in 7 cases out of 68. Correction of this fault leads to an immediate improvement in the electroencephalogram.

It may be argued here that the high venous pressure reading will give the indication that the superior vena cava is obstructed.

High venous pressure

Cerebral perfusion is obviously dependant not only upon arterial pressure but also upon the venous pressure. For instance, with an arterial pressure of 60 mm. of mercury and a venous pressure of 5 mm. of mercury the electroencephalogram is often quite good. However, with the same arterial pressure and a venous pressure of 20 mm. of mercury the electroencephalogram has shown a lot of slow frequency large amplitude activity but the high venous pressure has not been due to obstruction of the superior vena cavae by the cannula. As well a perfectly normal electroencephalogram has been seen with a venous pressure of 25 mm. of mercury and an arterial pressure of 95 mm. of mercury.

Cerebral vasoconstriction

Active or passive hyperventilation with the washing out of carbon dioxide invariably leads to a worsening of the electroencephalogram. Sugioka and Davis (1960) have shown in dogs that hyperventilation with either room air or 100 per cent. oxygen there is a fall in the partial pressure of oxygen in cerebral tissues from a normal figure of 8-15 mm. of mercury down to 3-5 mm. of mercury. They have also shown an excellent correlation between low alveolar carbon dioxide and a low cerebral oxygen partial pressure. These workers have demonstrated a dramatic rise in cerebral oxygen tension content when carbon dioxide is added to the inspired gases during hyperventilation and the prompt fall to low levels when the carbon dioxide is removed.

Darrow (1945) observed photometrically through a cranial window in a cat's skull, the blanching of pial vessels, indicating vasoconstriction when the animal was hyperventilated and flushing of the vessels, indicating vasodilatation when carbon dioxide was given. Therefore, with hyperventilation, production of slow waves of large amplitude is not an indication of deeper anaesthesia but rather hypoxia secondary to cerebral vasoconstriction.

Hypoxia

Hypoxia certainly causes changes in electroencephalogram but as oxygenation is monitored very closely it is true to say that very few changes are due to lack of oxygen. Clowes and associates (1953) investigated hypoxia in anaesthetized dogs and found no effect on the electroencephalogram until the haemoglobin of arterial blood reached 40 per cent. saturation with oxygen. While this may be true in dogs, with critical clinical cases cerebral tissues are more sensitive to oxygen lack. It has been found on several occasions after a moderately long perfusion that there is a slight retrogression in the electroencephalogram as evidenced by the appearance of a few slow waves. Invariably, there has been some defect in the oxygenator, e.g. bridging of the blood in between discs or a fairly high bubble formation and yet the polarograph has shown 80-85 per cent. saturation.

Hypothermia

With progressive reduction in temperature changes in the electroencephalogram are noted between 31°C and 29°C in about 50 per cent. of patients. This change is made up of a small amount of slowing but not much variation in amplitude. Below 29°C the frequency tends to be reduced more frequently than above this temperature. The tracing does not become isoelectric until about 20°C ± 2°C pharyngeal temperature and reappears on rewarming at about the same level.

However, any other change such as deeper anaesthesia, hypoxia, etc., is additive with hypothermia and tends to cause deterioration of the electroencephalogram. Following circulatory occlusion there is often a delay before return of electroencephalographic activity. In this situation hypoxia is being added to hypothermia.

The first sign of activity noted is small voltage fast frequency (approximately 5 microvolts usually in excess of 15 cycles per second). The finding of this "file" pattern after a period of hypoxia is said to indicate

poor prognosis, but the experience at the Alfred Hospital is that it is an invariable finding following hypothermia and circulatory occlusion.

The electroencephalogram can often be long delayed in its return; again said to carry a poor prognosis. One patient for aortic valvotomy who was submitted to deep halothane, hypothermia at 30°C and circulatory occlusion for 8 minutes showed no sign of return of electroencephalogram after 1 hour. However, he showed no mental deterioration on wakening.

CONCLUSION

This paper presents a discussion of some of the factors affecting the electroencephalogram during cardiac surgery.

The electroencephalogram has for the anaesthetist two main uses — an indicator of depth of anaesthesia and a valuable aid in monitoring the perfusion of the tissues most sensitive to oxygen lack.

ACKNOWLEDGEMENTS

It is a pleasure to record my thanks to Dr. R. H. Orton for his invaluable advice and criticism in the preparation of this paper.

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A CASE OF ATTEMPTED REPAIR OF THE SUPERIOR MESENTERIC VESSELS*

By W. Brian Fleming

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THE small branches of the superior mesenteric vessels are frequently ruptured in crush injuries and gun-shot wounds of the abdomen and the management of these cases by ligation of the bleeding points and perhaps resection of an avascular loop of small bowel is familiar to all. Our experience of damage to the main trunks of these vessels however, usually is confined to moribund patients who have survived shock and haemorrhage from multiple intra-abdominal injury only long enough to reach hospital. The vascular lesion is then just one of many irreparable injuries. Rupture of the main superior mesenteric vessels alone is rare and there is no extensive literature describing the results of their ligation or repair. Ulverstad (1954) stated that he could not find a single report of repair of the superior mesenteric vessels in the war literature. No surgeon is sure of what will happen, if anything, if he ligates the superior mesenteric vessels at various levels in an otherwise healthy young patient, although he might reasonably guess that at least some of the bowel might die. One cannot use the experience gained from study of cases of superior mesenteric vascular occlusion nor even from cases in which the intestinal blood supply has been examined before and after superior mesenteric embolectomy because in most of these patients both the vessels and the general state of the circulation are abnormal.

Only two cases of repair of injuries to the superior mesenteric vessels have been reported in the last ten years. Kleitsch, Connors and O'Neill (1957) described a 19-year-old man in whom a 0.22 calibre bullet had almost divided the superior mesenteric artery and vein just below the level of the transverse mesocolon. A haematoma of the base of the mesocolon and small bowel mesentery was seen, but not explored and the abdomen was closed. Ten hours later the abdomen was reopened because of continuing intra-abdominal haemorrhage and on this occasion there was blood in the peritoneal cavity. The peritoneum over the haematoma was incised and blood spurted from the superior mesenteric artery. Bleeding was controlled and then the vein was sutured and the damaged segment of superior mesenteric artery excised and an end-to-end anastomosis was performed. Presumably despite the damage to the main vessels the collateral circulation to the intestine was adequate at the time of the first laparotomy. The patient recovered and the bowel was shown intact at a third laparotomy on the 7th post-operative day.

Ulverstad (1954) reported a case in which a man was crushed between a truck tail-gate and a loading platform. He had a laceration in the posterior peritoneum 3 cm. below the ligament of Treitz and beneath it was a ragged laceration of the superior mesenteric artery below the level of the middle colic artery. When the bleeding had been controlled, the bowel from just below the ligament of Treitz to the mid-transverse colon was without blood supply but it was restored after suture of the artery and removal of the clamps. No anti-coagulant was used post-operatively. The patient recovered although diarrhoea persisted for several weeks post-operatively.

Case History

A 27-year-old man was admitted to the Footscray and District Hospital on 9th April, 1960, shortly after being shot in the abdomen with a 0.22 calibre bullet. He was transfused and an immediate laparotomy was performed. The track of the bullet was traced from an entrance wound just above and to the right of the umbilicus, through the anterior wall and greater curve of the stomach and then through the base of the transverse mesocolon just to the left of the middle colic vessels. A very large haematoma expanded the base of the small bowel mesentery below the mesocolon and on incising this bright blood spurted from several places. The vessels were exposed by gently kneading the base of the mesentery between the left index and thumb displacing the

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blood clot and fat. After controlling the haemorrhage with clamps the state of superior mesenteric vessels was found to be as illustrated in Fig. I. There was a lateral tear 1 cm. long on the right side of the artery immediately below a minute branch running to the right which was divided. No jejunal branches were seen arising opposite the tear. The front and back walls of the vein were perforated just above the confluence of three major branches. The state of the small bowel prior to this was not noted owing to the urgent need to control the bleeding, but with clamps applied to the vessels well above and below the lacerations the small bowel was cyanosed and immobile. With the artery clamped alone however, there was still a considerable flow of blood from the distal cut end of the vein.

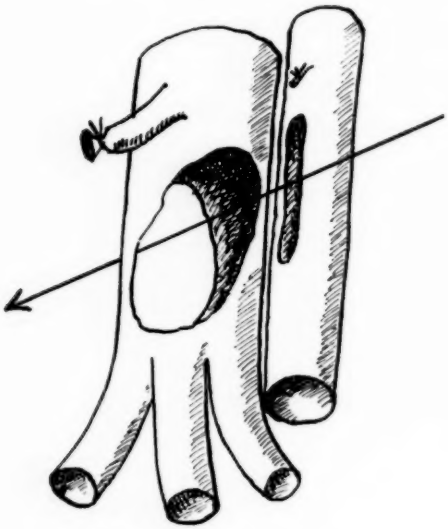

FIG. I. A diagram showing the injury to the superior mesenteric vessels, a linear tear in the artery and perforation of the anterior and posterior walls of the adjacent vein.

The tear in the artery was repaired with a continuous suture of 00000 "Deknatel." The intact, but shredded right wall of the vein was then divided and trimmed leaving a deep wedge shaped defect (Fig. II) which was then sutured with the same material. On releasing the clamps the circulation and pulsation returned very briskly and the front wall of the vein distal to the suture line split longitudinally and had to be repaired with interrupted sutures. Inspection of the small bowel mesentery now showed good peripheral pulsation everywhere along its length. No attempt was made to explore further and remove the bullet as it was thought to lie away from the aorta and inferior vena cava.

The patient received 6 litres of blood during and after the operation. His convalescence was quite uneventful and at no time did he become distended. Bowel sounds returned after three days and oral feedings were then commenced. He remained well except for a mild wound infection and did not develop diarrhoea. Anti-coagulants were not given. On 26th April he was transferred to the Royal Melbourne Hospital where an aortogram was performed by Dr. W. S. C. Hare to check the relation of the bullet to the aorta and also to demonstrate the state of the intestinal circulation. This showed a complete block of the superior mesenteric artery at the site of the repair and no filling of the vessel below, but at least 2 large jejunal branches running to the left above the level of the block were shown (Fig. III). When seen two months after operation the patient was well and was about to return to work. He was not anaemic and was putting on weight. He had had one short episode of diarrhoea lasting two days just after leaving hospital but his motions were normal after that.

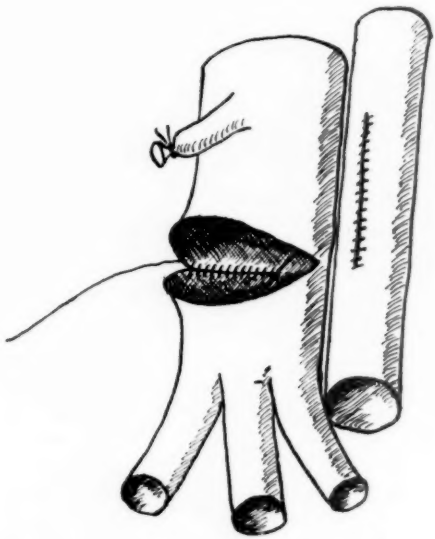

FIG. II. A diagram showing the method of repair of the torn superior mesenteric artery and vein.

DISCUSSION

If the superior mesenteric vessels are occluded no site could be more favourable for complete survival of the bowel than just below the origin of the first 2 or 3 jejunal branches. At this level of obstruction the potential collateral circulation is greatest. In our case pulsation was observed across the repaired arterial segment as soon as the clamps were removed. The patient's convalescence was rapid and straightforward and

the result attributed to a successful repair of the vessels. It was not until an aortogram was performed that it was realized that occlusion of the main trunk had occurred. Late aortograms were not performed in Ulverstad's (1954) nor in Kleitsch *et alii* (1957) cases and it is possible that they too were deceived by the clinical state of their patients into believing that the repaired vessels had remained patent.

FIG. III. A photograph of an aortogram performed two and one-half weeks after the injury showing a complete block of the superior mesenteric artery at the level of repair. At least 2 and possibly 3 jejunal arteries can be seen arising from the left side of the artery above the block.

In retrospect two observations made in the case being reported suggest that the collateral circulation was adequate at the time of the repair.

(a) The bowel was not obviously avascular before attempts to control the haemorrhage were made, although no particular attention was paid to the colour of the intestine at that stage. It is likely however, that if the bowel had been completely avascular this state would have been recognized immediately the abdomen was opened.

(b) Some time after the artery was occluded with clamps the flow from the distal end of the lacerated vein was still profuse.

Kleitsch *et alii* (1957) apparently thought the colour of the bowel was normal at the time of the first laparotomy in their case again suggesting that the collateral circulation was adequate. Only continued intra-abdominal haemorrhage forced them to re-open the abdomen, explore the haematoma and repair the artery.

One wonders what might have happened if the vessels had been ligated in all 3 patients instead of subjecting them, while in a critically ill state, to a long and difficult vascular repair, especially when there were other lesions with which to deal. Ulverstad (1954) had definite indications to repair the artery in his case however. After the bleeding had been controlled by ligatures it was then noticed that the bowel was avascular from the ligament of Treitz to the mid-transverse colon. The superior mesenteric vessels were then carefully exposed and it was found that one of the previously applied ligatures encircled the superior mesenteric artery, occluding it at the site of a laceration. Division of the ligature and suture of the laceration was followed by complete restoration of the blood supply.

The fact that the avascular length of bowel included the right half of the transverse colon (usually supplied by the middle colic artery) suggests some cause for the depleted blood supply in addition to the ligation of the superior mesenteric artery below the expected level of origin of the middle colic artery. The tear in the posterior peritoneum in Ulverstad's (1954) case was 3 cm. below the ligament of Treitz and the accompanying diagram in the paper shows the presence of at least one jejunal branch above the level of the ligature occluding the superior mesenteric artery. One would have to postulate a vascular abnormality, occlusion of other main vessels by ligatures, arterial spasm and/or temporary peripheral circulatory insufficiency to explain adequately the avascularity of the transverse colon. The circumstances described in Ulverstad's (1954) case, therefore, do not contradict the suggestion that it is probably

safe to ligate the superior mesenteric vessels if the lesion is below the level of origin of the first 2 or 3 jejunal branches. Certainly in future cases it would be wise to place atraumatic clamps over the bleeding points on the main vessels, making sure the collaterals were not occluded, and to repair the vessels only if the peripheral pulsation was absent still after restoration of the blood volume.

This case demonstrates that the collateral circulation by-passing an occluded superior mesenteric artery below the level of the first 2 jejunal branches in an otherwise healthy patient can be sufficient to maintain viability and apparently normal function of the small bowel.

SUMMARY

1. A case is reported of repair of the superior mesenteric vessels lacerated by a bullet. Although the patient recovered without subsequent gangrene of the bowel an aortogram showed complete occlusion of the artery.
2. Two previously reported cases of other authors of successful repair of lacerations at a similar level are described.
3. It is concluded that simple ligation rather than repair of lacerations of the superior mesenteric vessels is probably safe provided that the lesion is below 2 or 3 intact jejunal branches of the artery.

ACKNOWLEDGEMENTS

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Books Reviewed

SHAW'S TEXTBOOK OF OPERATIVE GYNAECOLOGY.

Revised by JOHN HOWKINS. Second Edition. Edinburgh and London: E. & S. Livingstone Limited, 1960. 11" x 8½", 484 pp., 428 figures. Price: £5 5s. (stg.).

The outstanding impression after reading this book, is one of satisfaction. Credit must be given to all associated with its production and presentation, both aspects reaching the pinnacle of the publisher's art.

The author not only contributes a fitting memorial to his teacher and friend but he gives to British gynaecology a book for which there has been a need for years. The master would be proud of his pupil.

Text books, no matter how well illustrated, or how excellent and detailed the text can never replace apprenticeship in the training of a surgeon. If this book were used by house surgeons and registrars during their training it would be an excellent guide because indications and techniques for operations recognized and proved by British gynaecologists are described in detail.

The author does not attempt to cover all aspects of the subject but rightly stresses the more common procedures. Perhaps in future editions he may be able to incorporate some of the statistics from his own extensive practice as well as those from his colleagues at St. Bartholomew's rather than rely on the results of others. The illustrations are clear and beautifully presented. The easy style of writing should make this book a favourite with both student and surgeon.

The chapter on abdominal conditions likely to be encountered by a gynaecological surgeon is valuable and together with a chapter concerning the use of radiation complete an excellent book.

TRANSACTIONS OF THE INTERNATIONAL SOCIETY OF PLASTIC SURGEONS—Second Congress, London, 1959.

Edited by A. B. WALLACE, M.Sc., F.R.C.S.Ed. Edinburgh and London: E. & S. Livingstone Limited, 1960. 10" x 7", xvii plus 589 pp., illustrated. £4 10s. (stg.).

The appearance of this volume, so accurately and beautifully produced in the year following the second International Congress of Plastic Surgeons, is a triumph for both the editor and the publishers. This record of the meeting held in London in 1959 is a splendid successor to the publication of the transactions at the first International Meeting in Sweden in 1955, and so wide has been the representation at these two Congresses and so extensive the coverage of all aspects of plastic surgery in the large number of papers presented, that one can regard these two volumes not only as works of great historical value but as presenting a wide and accurate cross section of plastic surgery throughout the world at the time of publication.

In any such work there must inevitably be good and bad material presented, material whose quality is undoubted and material which excites controversy and criticism, and it is so with this book. Notwithstanding this, the selection committee responsible for organizing the programme did its task admirably. There are sections on surgery of cleft lip and palate, anaesthesia, surgery of the face, cancer surgery, maxillo-facial and jaw surgery, hand surgery, skull replacement surgery, and the reconstruction of ears, genitalia, eyelids and eye-sockets. There are other sections on the use of homografts, the management of burns, on cosmetic surgery, the surgery of specific infections and miscellaneous subjects. In short there is no branch of the specialty which is not the subject of one or more papers from various centres throughout the world.

The book is a large one but not a particularly expensive one in relation to its contents. It will be of intense interest to all plastic surgeons, and to many others besides who are interested in some particular aspect of the specialty.

THE EYE IN GENERAL PRACTICE.

By C. R. S. JACKSON, M.A., B.M., B.Ch.(Oxon.), D.O.M.S., F.R.C.S.Ed. Second Edition. Edinburgh and London: E. and S. Livingstone Limited, 1960. 8½" x 5½", vii plus 152 pp., 26 illustrations. Price: 21st. (stg.).

This small volume deals in an adequate manner with ophthalmic conditions commonly seen in general practice. It fulfils the aims the author has set out in the preface to the first edition. These are, to describe the common diseases of the eye, to enable the practitioner to recognize severe ocular disease — and so seek specialist advice, and finally to understand more fully hospital reports.

The subject matter is divided into three sections; the diseases of the eye; ophthalmic manifestations of systemic disease; a section concerning the welfare of the blind, and the British National Health Ophthalmic Service.

The section in diseases of the eye follows the usual pattern, dealing with conditions of the lids, conjunctiva and cornea, cataract and glaucoma, and on the whole gives a well balanced account.

Diseases of other parts of the body which have ocular manifestations include cardiovascular disease and intracranial, endocrine and metabolic diseases.

This is a book that can be recommended to those engaged in general practice and will prove of value to medical students and resident medical officers.

One disappointing feature is the illustrations. Although mainly in colour, in many, the actual lesion to be demonstrated is so small in the photographs as to be unrecognizable.

OBSTETRICAL AND GYNAECOLOGICAL PATHOLOGY

By R. E. REWELL. Edinburgh and London: E. and S. Livingstone Limited, 1960. 8" x 5½", 435 pp., 150 illustrations. Price: 50s. (Stg.).

This book is intended for the post-graduate student studying gynaecology and obstetrics as a clinical subject and reading for a higher examination. It is not a practical manual, and gives the reader a theoretical basis for his clinical work. The author is a morbid anatomist at the Liverpool Maternity Hospital and the Women's Hospital, Liverpool, and the book contains his experiences gained

at these hospitals during a decade. His approach is much wider than this, as he was a Professor of Pathology in Madras under the Colombo Plan.

The gynaecological section deals with the menstrual cycle, functional disturbances of the endometrium, tumours of the endometrium and myometrium, diseases of the cervix, vagina, vulva, Fallopian tube and ovary, endometriosis, tuberculosis and cytology. The obstetrical section deals with diseases of the placenta, fertility studies, maternal mortality and morbidity, defects in blood clotting, anaemia in pregnancy, iso-immunization, puerperal infections and peri-natal mortality.

Each subject is adequately dealt with from the pathological point of view, the author's views on controversial matters being fairly stated. The illustrations are very good, in particular the photomicrographs with the arrows pointing to features of interest. Excellent references are given at the end of each chapter.

This book is essential for every post-graduate studying for membership of the Royal College of Obstetricians and Gynaecologists, and will give assistance to the pathologist or surgeon with general experience in pathology but limited acquaintance with obstetrics and gynaecology.

CIRCULATORY ULCERS — A PHYSICAL APPROACH.

By HILTON G. TRANCHELL and CHARLES R. BANNISTER. Bristol: John Wright and Sons Limited, 1960. 7½" x 5", 91 pp., 16 figures.

This is a small, easily read book of only 91 pages in which an endeavour has been made to briefly cover a very large subject. The first chapters describe the types of ulceration and, unfortunately, the aetiological appearances are not clearly depicted in the microphotographs, but, specific attention is drawn to the frequent contaminants of ulcers and also, the significance of the site, especially in pointing out that arterial ulcers occur in different sites to venous ulcers.

The aims and technique of treatment are clearly set out, specifically mentioning the important adjunct to treatment of physiotherapy in relieving pain, mobilizing the joints, re-educating the muscle and improving the circulation.

Local preparations are divided into toilet solutions, skin protectives and dressing solutions. It would, however, do well to mention that the excessive use of most preparations, in time, will produce inflammatory reactions of the skin surrounding the chronic ulcers.

The last chapters are devoted to the treatment, firstly outlining in detail the routine of local physiotherapy and, secondly the use of skin grafts. It does not, however, mention the necessity of dealing with the underlying veins if the graft is to remain stable. It merely advises the use of elastic support.

This is a small, easily read book which outlines briefly our basic concept of the assistance that physical medicine can give us in handling chronic ulcers of the lower limbs and should be commended to all medical practitioners and physiotherapists.

NEOPLASTIC DISEASE AT VARIOUS SITES.
Volume III — Cancer of the Rectum.

Edited by CUTHBERT E. DUKES. Edinburgh and London: E. and S. Livingstone Limited, 1960. 10" x 7", 295 pp., 82 illustrations. Price: 50s. (stg.).

A series of monographs on neoplastic diseases at various sites has been prepared under the general editorship of Professor D. W. Smithers. *Cancer of the Rectum* is the third of these and none is better fitted for the task of its preparation than Dr. Cuthbert Dukes. Cuthbert Dukes was appointed pathologist to St. Mark's Hospital in 1922 and much of the international fame of this Institution is directly due to his work. The clarity of his thinking and the logic of his reasoning have brought recognition from all over the world. The reader of this volume can feel his influence from the outset.

The first section of the book concerns the mortality from the disease. Three fascinating chapters and an appendix have been compiled by Dr. R. A. M. Case. He supports all his statements with facts and impressions. He shows that the mortality is slightly greater in the poor class of the community. He was unable to show that any occupational hazard, such as can be demonstrated with cancer of the bladder, existed. It is interesting to observe the low mortality from the disease in Italy and Japan, and the high mortality in Denmark, Netherlands, New Zealand and the United Kingdom.

Dr. Cuthbert Dukes, in the second section, deals with predisposing factors and the control of the development of carcinoma of the rectum. In typical style, he surveys the facts which are known concerning the aetiology (it is interesting to note that the spelling "etiology" rather than "aetiology" is used).

The third section is concerned with pathology. Even large bowel tumours in domestic, laboratory and wild animals receive consideration. Dr. Dukes has subdivided his group B carcinomata into those with minimal spread, those with moderate spread, and those with extensive spread. Nearly 90 per cent. of the former group survive five years as against 57 per cent. of the latter. He emphasizes again the serious prognostic significance of established lymphatic spread. Dr. Basil Morson, who has succeeded Dr. Dukes at St. Mark's Hospital, has given a lucid description of the squamous cell tumours around the anal region. The comparative infrequency of this tumour is demonstrated by the fact that in a period of twenty-eight years only 157 cases have been treated at St. Mark's Hospital.

Section four deals with anatomy and with the clinical features. These are always difficult parts about which to write, but have been completed here in good style. Bussey and Wallace outline the age, sex and site, Parks the anatomy, and Muir the clinical diagnosis. Mr. W. B. Gabriel discusses biopsy.

Section five is devoted to the treatment of rectal cancer. Professor Goligher traces the development of surgical treatment for this disease and then leaves the leading exponents of the various types of operation to discuss them more fully. The Miles abdomino-perineal excision is described by Mr. Lawrence Abel, the perineo-abdominal excision by Mr. W. B. Gabriel, the synchronous combined excision by Mr. Lloyd Davies, the restorative excision by Mr. Naunton Morgan. There are sections on

malignant polyps by Mr. Lockhart-Mummery and radiotherapy by Dr. I. G. Williams. Pull-through procedures are criticized and are not described in detail, but there is no doubt that patients prefer an anal opening, even if it is not perfect. If there is any criticism to be made of this book, it is perhaps the omission of this method of restorative resection, performed so well in the United States by Bacon, Turnbull and others.

Post-operative care and complications are outlined in section six by Henry Thompson, Ian Todd and Gordon Ungley. And, finally, in section seven of the book, Bussey, Dukes and Lockhart-Mummery analyse the results of treatment at St. Mark's Hospital.

This book is quite short, but it contains a very complete survey of the problems created by carcinoma of the rectum. Although 18 different authors have combined, the chapters flow evenly, without repetition and Dr. Dukes is to be congratulated on the magnificent way he has organized his team. A monograph bearing his name is never brushed aside, and this one is no exception, and more so because the "batting order" includes so many names with world-wide reputations.

VARICOSE VEINS — A PRACTICAL MANUAL.

By R. ROWDEN FOOTE. Third Edition. Bristol: John Wright and Sons Limited, 1960. 9½" x 6½", xx plus 356 pp., 304 figures. Price: 63s. (stg.).

This is the third edition of a practical manual on the anatomy, aetiology and management of varicose veins and ulcers. This edition has been revised and now includes some of the newer concepts of anatomy and management. It seems a pity that in the first historical chapter the author states he has not altered this chapter and that he has not included details of contemporary thought regarding the anatomy of perforating veins. However, this modern concept of perforating veins is dealt with in other sections of the book.

The anatomy and physiology has been revised to include details of the perforating veins and it is noted that the importance of the calf muscle pump in maintaining the venous return from the lower extremity is stressed.

In the surgical treatment of varicose veins it has long been accepted that the results depend on adequate pre-operative assessment and it is noted that this aspect is stressed in order to determine which patients should be treated by surgical means or by conservative methods. Good results in varicose vein surgery can only be obtained by adequate pre-operative preparation and mapping of the veins in order to plan the procedure. The posturing of the patient on the operating table in order to promote haemostasis and the placing of incisions in Langer's lines in order to minimize tension of the skin edges is mentioned.

Another important point in the book which the author brings to the reader's attention is that, in stripping, if too small a stripper is used, the veins may be invaginated, torn off and inadequate stripping obtained.

In dealing with the perforating veins the author recommends that small incisions only be made over the site where the perforators are detected clinically. In many cases such small incisions are adequate but very frequently if this method is employed perforating veins will be missed, especially as they cannot always be mapped with the accuracy that is often desired and a longer more adequate incision over the greater part of the lower third of the calf will overcome this difficulty.

In the management of cases by conservative means attention is drawn to the use of stockings and to correct posturing whenever possible. The use of sclerosants is also mentioned and in particular it is drawn to the attention that nowadays the most frequent use of this method of treatment is during the post-operative follow-up stage after stripping.

In the treatment of ulcers by compression mention is made of the various methods of compression, either by elastic bandaging or elastoplast. Unfortunately the danger of allergic responses and dermatitis to some of these methods is mentioned but not stressed and very little space (two pages) is given to the use of one way stretch bandages which form such an important method of management in present-day treatment.

In the surgical management of ulcers the author has obviously encountered the difficulties of sub facial ligations, in which the wound breaks down, the area gapes and he draws attention to the fact that sub facial ligations are not required very frequently, if adequate control of oedema and improvement in the induration is obtained by simple conservative measures pre-operatively.

This is an excellent book which has been revised in order to include some of the modern concepts since the importance of perforating veins has been brought to our notice. Unfortunately in the greater portion of the book the older well-tried methods are included in detail while some of our more recent advances which are accepted and which comprise the greater part of the modern treatment are not given in sufficient detail.

TREATMENT OF CANCER AND ALLIED DISEASES.
Volume 4 — Tumours of the Breast, Chest and Oesophagus.

Editors: GEORGE T. PACK and IRVING M. ARIEL. New York: Paul B. Hoeber, 1960. 10" x 7", 667 pp., 650 illustrations. Price: $30.

The volume contains in its first section eleven chapters dealing with the treatment of tumours of the breast and among these are "Radical Mastectomy in Continuity with Internal Mammary Lymph Node Dissection," by Jerome A. Urban; "Post-mastectomy Lymphedema of Upper Extremity," by James M. Winfield, Walter L. Mersheimer and Martin E. Silverstein; "Hormonal Therapy of Mammary Carcinoma," by B. J. Kennedy; "Treatment of Metastatic Breast Cancer by Hypophysectomy," by Bronson S. Ray and Olof H. Pearson, and other equally arresting contributions. All are of particularly high standard, presented with the utmost clarity and detail and with a keenly analytical objectivity.

Twenty-two contributions on intrathoracic tumours form the second section. The choice of subjects is very satisfying, such as "Pulmonary Cancer: An Appraisal" (Irving M. Ariel and George T. Pack); "Surgical Treatment of Metastases to the Lung" (Hiram T. Langston); "Pre-operative and Post-operative Management in the Treatment of Neo-

plasms of the Chest" (John H. Gibbon, Jr., and Thomas F. Nealon, Jr.) and "Treatment of Tumours of the Thymus" (O. Theron Clagett and John R. McDonald). It is perhaps surprising that there are three separate chapters on irradiation and none on chemotherapy.

Perhaps the third section on tumours of the oesophagus is the most attractive and challenging — seven essays all admirably presented, the first four dealing in meticulous detail with the operative problems, models of clarity and of the highest standard, the last three devoted to problems and methods of radiation therapy.

In the dedication to the memory of William S. Halsted and Evarts A. Graham are these words: "They are the exemplars of the ideal surgeon as characterized by Guy de Chauliac, the master surgeon of Montpellier, in his extraordinary book *Grande Chirurgie*." This volume continually speaks of *Grande Chirurgie*.

ANATOMY AND PHYSIOLOGY FOR RADIOGRAPHERS.

By J. E. BLEWETT, M.D., D.M.R., F.F.R., and A. M. RACKOW, B.Sc., M.B., D.M.R.E. London: Butterworth & Co., 1960. 8½" x 5½", 322 pp., 22 plates, 133 illustrations. Price: 52s. 6d. (stg.).

This book, which is making its first appearance, is rather ambitious. Although not indicated by the title, it includes brief mention of the pathology medicine and surgery of several conditions. In so doing, the authors reflect the modern tendency to include some knowledge of these subjects in the teaching of radiographers.

The subjects of anatomy and physiology are adequately covered, although the anatomy is rather brief, and could be expanded with advantage. This applies particularly to surface anatomy, a sound knowledge of which is so essential to diagnostic and therapeutic radiographers alike.

An increase in the number of diagrams would be an improvement. Generally, the diagrams are adequate, some being excellent. Others, however, are too small and lacking in detail and there are a few errors in terminology. The use of a second colour in some diagrams adds to their clarity. The inclusion of plates showing normal radiographic appearances with accompanying diagrams is a good feature. Students will also appreciate the inclusion at the end of each section of a list of relevant diseases and medical terms.

The book will fill a long-felt gap in the education of radiographers and will add to their knowledge. Its circulation however will be confined to those who have not had a medical training and are seeking a basic knowledge of these subjects.

MODERN TRENDS IN DIAGNOSTIC RADIOLOGY.

Edited by J. W. McLAREN. London: Butterworth & Co., 1960. 10" x 7", 274 pp., 209 illustrations. Price: 102s. 6d. (stg.).

This is the third volume in the series. It makes a welcome appearance, it being seven years since its predecessor was published. Diagnostic radiology is making such strides at the present time, that it is to be regretted that "Modern Trends" does not appear more frequently.

The editor has chosen well recognized leaders in each field to contribute. The several chapters on the various techniques using contrast media in the cardio-vascular system are written almost entirely by Scandinavian authors. This is a tribute to the vast amount of research, constant progress, and excellent results they have achieved.

The opening chapter describes some of the recent advances in equipment. Here the absence of mention of the use of the television screen is a surprising omission. Mention is made in several chapters of the uses of cine-radiography and its application, particularly in the fields of investigation of the cardio-vascular and urinary systems and of the oesophagus.

A brief chapter is devoted to protection, a subject which is also discussed in the chapter on obstetric radiology. Here it will now be generally agreed with the author that "ante-natal radiology, carefully conducted, has far more to offer in the immediate and ultimate interests of mother and child than the deliberate withholding of radiological examination because of radiation risks."

Many radiological procedures are dealt with, including the urinary and biliary tracts, soft tissue radiology and rare conditions of bone. The reproductions throughout are of a uniformly high standard.

Finally there is a useful chapter on the various agents used as contrast media Their advantages and disadvantages are discussed and the media of choice for the various examinations indicated. The need for new and improved media is discussed and their desiderata pointed out.

A wide field has been well covered in a short book. Many subjects therefore are only briefly discussed. A good bibliography however is adequate compensation for this brevity. An excellent index is included. Radiologists will find this volume a welcome addition to its predecessors.

SURGERY OF THE ACUTE ABDOMEN.

By J. A. SHEPHERD. Edinburgh and London: E. and S. Livingstone Ltd., 1960. 10¼", x 6½", 1,228 pp., 43 plates, 11 figures. Price: £5 (stg.).

Mr. Shepherd has kept a detailed account of 1,179 abdominal emergencies operated upon personally while holding successive registrar and consultant appointments. These form the basis for this book.

The contents of the book have been arranged in an orthodox manner. Chapters on the principles and methods of diagnosis are followed by the principles of management. After this opening, acute emergencies of the various viscera are exhaustively surveyed. Mr. Shepherd has created a fine balance by quoting his own cases, by his observations on others, and by his reference to the literature. The common lesions are thoroughly reviewed, whilst few rare conditions have been omitted.

The section on perforated ulcer is perhaps the most complete review published on this important subject. On the management of large bowel obstruction caused by carcinoma there could be some cause to dispute the author's views which are based on a personal series of 23 cases treated over a period of eight years. Nevertheless, the opinions presented are sound and orthodox. This applies throughout the book.

The book is sparsely illustrated, but the figures have been chosen carefully. E. & S. Livingstone have published the book in typically excellent style.

This publication is an important one for surgeons who have just acquired their Fellowship and who are so often asked to cope with emergency surgery without any further opportunity for training and it can be firmly recommended as a reference book for those wishing to obtain a fair review of current opinion on acute surgical emergencies, and there will be few conditions encountered by the surgeon to which the author has not given some consideration.

ACTIVE ALERTED POSTURE.

By W. E. TUCKER. First Edition. Edinburgh and London: E. & S. Livingstone Ltd., 1960. 8½" x 5¼", 64 pp., 24 illustrations. Price: 10s. 6d. (stg.).

The author states that those who continually adopt a slumped posture tend to develop bodily ills which might be avoided by adopting the "active alerted posture." The body then is in a state of preparedness when body activity is contemplated. Thereby there would be a reduction of accidents in industry or sport and also in later life warding off tissue degeneration, such as osteoarthritis. In the perfect active alerted posture, the body is held upright with the knees slightly flexed, "navel press-buttoned to spine," buttocks "pinch proof," shoulders lifted and bent slightly forward and "top of head trying to touch the ceiling." Thereafter "a person proceeding into motion is then like a arrow shot from this bow." The small book is clearly written and the simple line illustrations are excellent.

A TEXTBOOK OF X-RAY DIAGNOSIS.

Volume 4. Bones, Joints and Soft Tissues.

By S. COCHRANE SHANKS and PETER KERLEY. Third Edition. London: H. K. Lewis & Co. Ltd., 1959. 10" x 7½", 714 pp., 735 illustrations. Price: £5 10s. (stg.).

The latest edition of this volume, which is devoted to orthopaedic radiology, follows closely the pattern of its predecessors. Many additions have been made to the text and in the number of radiographs which have been increased by almost 200.

The book is splendidly produced and the quality of the reproduced radiographs is uniformly good. However the continued use of positive reproductions rather than the negative, to which both radiologists and clinicians are accustomed, remains a criticism.

This volume is now becoming almost too large and perhaps consideration could be given to omission of the chapter on general pathology of bone which may well be left to books on that subject.

The sections which have been most enlarged are those on hyperparathyroidism, renal osteodystrophy and on bone and cartilage tumours. In the latter, two tumours, the chondroblastoma and the aneurysmal cyst of bone, previously described as giant cell variants, are now correctly classified, described and illustrated. Several excellent new radiographs have been included in this section. Chondrosarcoma has been described separately from osteogenic sarcoma, which, as the author states, cannot always be made, but is justified and should be attempted.

A chapter on plasma cell myeloma and multiple myeloma is a welcome addition. Changes occurring in the soft tissues, so important in early osteomyelitis, have now been included.

The only real criticisms which can be made are a few minor omissions, and a very small proportion of reproductions are not up to standard.

The book maintains its position as the foundation stone on which students of radiodiagnosis will base their learning of orthopaedic radiology.

SURGERY IN WORLD WAR II — NEUROSURGERY.

Volume II.

By MEDICAL DEPARTMENT, U.S. ARMY. Edited by R. GLEN SPURLING and BARNES WOODHALL. Washington: Office of the Surgeon-General, Department of U.S. Army, 1959. 10" x 7", 705 pp., 283 illustrations. Price: $7.00.

The second volume records the experiences of neurosurgeons of the U.S. Army Medical Corps in the management of spinal and peripheral nerve injuries. In the chapters on peripheral nerve injuries, Dr. Woodhall deplores the fact that there was no readily available guide for the administration and management of these problems at the onset of the War. Dr. Harvey Cushing had laid down general principles for the management of head injuries in World War I which were carried over into World War II. There was no comparable, useful guide for spinal and peripheral nerve injuries. Perhaps if there had been, a greater time lag would have occurred in changing over to the greater range and freedom of operative procedures following the much greater control of sepsis which was obtained in World War II by virtue of the introduction of penicillin and to the much better organization of evacuation of patients.

Nevertheless the volume does provide an excellent guide and reference book for this aspect of war surgery and it covers in detail much that can be applied in peacetime to the management of spinal and peripheral nerve injuries. There is less administrative detail in this volume and the clinical aspects are dealt with clearly, although on occasions the various authors of separate sections make statements which are mildly contradictory. One defect in the volume, which was also observed in the previous one, is that the information recorded is experience in isolation. Little or no reference is made to the organization or the results of other surgeons obtained before the United States entered the war.

The magnitude of the task of dealing with 1,500 paraplegics is perhaps reflected in some of the terminology. A centre for paraplegics is no longer called a unit but a paraplegic colony in which more than 100 patients were being rehabilitated at one time.

The three chapters dealing with the management of the acute compound spinal injuries, the urological problems and the routine care of spinal injuries, set out the general principles governing the management of this condition. These have, for the most part, obtained world-wide recognition and approval although there are some sharp differences of opinion about the use of the Stryker frame and the methods of bladder drainage. In particular, the acceptance of suprapubic drainage under certain conditions would be disagreed with by some British workers in this field. It is claimed that 70 per cent. of the

paraplegics passing through these centres were ambulatory at the end of their period of rehabilitation and training, but no adequate follow-up statistics are provided to ascertain how many of these persisted in attempts to walk with crutches after leaving hospital.

The discussion on intervertebral disc surgery is perhaps rather defeatist in attitude for it admits the futility of trying to get the majority of Service personnel with this condition back to anything approaching full and free activity.

The greater portion of the volume concerns peripheral nerve injuries, the Army organization during the years of conflict and the work of the Peripheral Nerve Registry are described and an outline of the documentation of cases is illustrated. Although the means of documentation of each case is set out clearly there is no chapter on the diagnosis of the peripheral nerve injury in the field, and it is felt that the inclusion of such information would have made the work more complete. The details of operative exposure and repair are very well set out although there has been some reduplication in separate chapters.

Causalgia provides material for another interesting chapter. Although no satisfactory theory is advanced for the origin of this strange condition, the relief of pain following sympathectomy is one of the major advances in peripheral nerve surgery obtained during World War II.

The neuro-pathological changes observed in nerve injuries are covered by excellent illustrations.

The book concludes with a series of appendices. The first group sets out instructions for the routine care of the paraplegic patient, while the second group comprises tables and statistics relating to recovery of peripheral nerve injuries.

The illustrations in this volume are, for the greater part, of good quality, but it is regretted that many of the diagrams and anatomical illustrations concerning the peripheral nerve injuries are so reduced in size that they lose much of their usefulness.

Finally, one is tempted to review this volume by comparison with the relevant chapters in the British Official War History and in some semi-official papers which were brought together in some special numbers of the *British Journal of Surgery*, but such comparison is unsatisfactory because there are differences in viewpoint and format. Although there is much that is redundant in this volume, it does cover all the aspects of spinal and peripheral nerve injuries very completely.

BODY FLUIDS IN SURGERY.

By A. W. WILKINSON, Ch.M., F.R.C.S.E., F.R.C.S. Second Edition. Edinburgh: E. & S. Livingstone Ltd., 1960. 8½" x 5½", 276 pp., 12 illustrations. Price: 21s. (stg.).

The first edition of this book was reviewed in this journal in 1959. This second edition has been enlarged by about one-third of the original. A large part of the content is unchanged but the section relating to the effects of injury has been rewritten and enlarged to advantage. A new chapter has been added, giving an account of the present knowledge of the important variations in composition and function in the infant with their bearing on post-operative management. Another new chapter has been written on the maintenance of chemical neutrality in the body. The description of "acid-base" balance has been largely rewritten.

As previously, the book provides the reader with a wealth of information culled from the very extensive literature of this complicated subject. The references given are excellent, but the general tone of the writing is that of a theoretical discussion for those who are interested to learn the bases of the subject rather than that of a textbook to which one can refer for a concise answer when faced with an urgent problem.

The introduction of the Bronsted-Lowry convention in regard to "acids" and "bases" is commendable although many readers will find the explanation unfortunately confusing.

The clinical applications of "acid-base balance" are not well explained and no clinical perspective is given to the more severe degrees of imbalance. In this regard, the book does not provide "down to earth" practical advice when it is most needed — i.e. in the severe and complicated emergency.

It is unfortunate in a book of this type that the problems of acid-base balance which may face the anaesthetist, are not given more prominence. If surgeons were more acquainted with the difficulties and limitations placed on the anaesthetist by some of these imbalances, it would make for better understanding between the two in difficult cases.

This is a useful book, provided the reader does not slavishly accept the clinical aspects. It does not provide accurate and concise answers to many severe problems where one may require the most assistance.

Books Received

AIDS TO SURGICAL ANATOMY.

By D. B. MOFFAT. Fifth Edition. London: Bailliere, Tindall and Cox Limited, 1960. 6¾" x 4", 220 pp., 54 illustrations. Price: 12s. 6d. (stg.).

INTRODUCTION TO DENTAL ANATOMY.

By J. H. SCOTT and N. M. B. SYMONS. Third Edition. Edinburgh and London: E. and S. Livingstone Limited, 1961. 8½" x 5¼", xi plus 388 pp., 239 illustrations. Price: 45s. (stg.).

CORRECTIONS

In the May issue of the *Journal* a review of "Arterial Embolism in the Limbs" was published on page 318. It was incorrectly stated that this was written by I. Dunsford and Jean Grant. The correct heading should have been:—

ARTERIAL EMBOLISM IN THE LIMBS.

By A. L. Jacobs, M.A., D.M. (Oxon), F.R.C.P. Edinburgh: E. & S. Livingstone Ltd., 1959. 10" x 6¾", xii plus 200 pp., 37 figures, 50 tables.

A paper by R. W. Beal entitled "Islet Cell Tumour of the Pancreas" appeared in the August issue of the *Journal*. The footnote "Supported in part by the National Health and Medical Research Council of Australia" should have referred to the Clinical Research Unit of the Royal Prince Alfred Hospital and not to the author.